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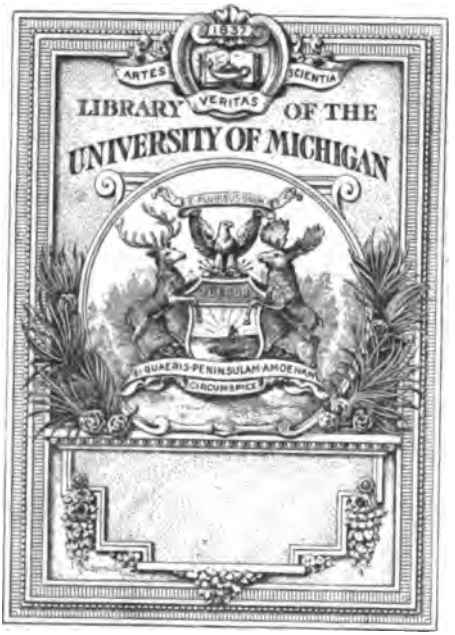

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TRANSACTIONS

OF THE

AMERICAN PEDIATRIC SOCIETY

TWENTY-SECOND SESSION

HELD AT THE NEW WILLARD HOTEL, WASHINGTON, D. C.,
MAY 3, 4 AND 5, 1910.

EDITED BY

LINNÆUS EDFORD LA FÉTRA, M.D.

VOLUME XXII.

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PRESIDENTS.

1889. A. Jacobi, M.D.
1890. J. Lewis Smith, M.D.
1891. T. M. Rotch, M.D.
1892. Wm. Osler, M.D.
1893. A. D. Blackader, M.D.
1894. John M. Keating, M.D.
1895. F. Forchheimer, M.D.
1896. Joseph O'Dwyer, M.D.
1897. Samuel S. Adams, M.D.
1898. L. Emmett Holt, M.D.
1899. Wm. P. Northrup, M.D.
1900. Henry Koplik, M.D.
1901. Wm. D. Booker, M.D.
1902. W. S. Christopher, M.D.
1903. J. P. Crozer Griffith, M.D.
1904. Augustus Caillé, M.D.
1905. C. G. Jennings, M.D.
1906. A. Jacobi, M.D.
1907. B. K. Rachford, M.D.
1908. C. G. Kerley, M.D.
1909. Charles P. Putnam, M.D.
1910. David L. Edsall, M D.
1911. Henry Dwight Chapin, M.D.

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Treasurer.....Charles Hunter Dunn, M.D.
Recorder and Editor.....L. E. La Fétra, M.D.

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John Lovett Morse, M.D.
S. McC. Hamill, M.D.
George N. Acker, M.D.
R. G. Freeman, M.D.
Alfred Hand, Jr., M.D.
L. E. La Fétra, M.D.

MEETING PLACES.

1888. Washington, D C. (Organization), September 18.
1889. Washington and Baltimore, September 20 and 21.
1890. New York, June 3 and 4.
1891. Washington, September 22 and 25.
1892. Boston, May 2, 3 and 4.
1893. West Point, N. Y., May 24, 25 and 26.
1894. Washington, May 29 and June 1.
1895. Virginia Hot Springs, May 27, 28 and 29.
1896. Montreal, May 25, 26 and 27.
1897. Washington, May 4, 5 and 6.
1898. Cincinnati, June 1, 2 and 3.
1899. Deer Park, June 27, 28 and 29.
1900. Washington, May 1, 2 and 3.
1901. Niagara Falls, May 27, 28 and 29.
1902. Boston, May 26, 27 and 28.
1903. Washington, May 12, 13 and 14.
1904. Detroit, May 30, 31 and June 1.
1905. Lake George, N. Y., June 19, 20 and 21.
1906. Atlantic City, N. J., May 30, 31 and June 1.
1907. Washington, May 7, 8 and 9.
1908. Delaware Water Gap, May 25, 26 and 27.
1909. Lenox, Mass., May 27 and 28.
1910. Washington, May 3, 4 and 5.
1911. Lake Mohonk, N. Y., May 31 and June 1 and 2.

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OFFICERS, 1911.

President.....Henry D. Chapin, M.D.
First Vice-President.....J. H. M. Knox, M.D.
Secretary.....Samuel S. Adams, M.D.
Treasurer.....Charles Hunter Dunn, M.D.
Recorder and Editor.....L. E. La Fétra, M.D.

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R. G. Freeman, M.D.
Alfred Hand, Jr., M.D.
L. E. La Fétra, M.D.
Isaac A. Abt, M.D.

MEMBERS.

Abt, Isaac A., M.D.....4326, Vincennes Avenue, Chicago
Acker, George N., M.D.....913, Sixteenth Street, Washington
Adams, Samuel S., M.D.....1, Dupont Circle, Washington
Baines, Allen M., M.D.....228, Bloor Street, Toronto
Blackader, A. D., M.D.....236, Mountain Street, Montreal
Bovaird, David, Jr., M.D.....120, West Fifty-eighth St., New York
Buckingham, E. M., M.D.....346, Marlborough Street, Boston
Caillé, Augustus, M.D.....753, Madison Avenue, New York
Carr, Walter Lester, M.D.....68, West Fifty-first Street, New York
Chapin, Henry Dwight, M.D.....51, West Fifty-first Street, New York
Churchill, F. S., M.D.....439, North State Street, Chicago
Coit, Henry L., M.D.....277, Mt. Prospect Avenue, Newark, N. J.
Cotton, A. C., M.D.....1485, Jackson Boulevard, Chicago
Cowie, D. M., M.D.....Lawrence Building, Ann Arbor, Mich.
Crandall, Floyd M., M.D.....113, West Ninety-fifth Street, New York
Dorning, John, M.D.....124, West Eighty-first Street, New York
Dunn, Charles Hunter, M.D.....220, Marlborough Street, Boston
Eaton, Percival J., M.D.....715, N. Highland Avenue, E. E., Pittsburgh
Edsall, David L., M.D.....1432, Pine Street, Philadelphia
Fife, Charles A., M.D.....1927, Chestnut Street, Philadelphia
Forchheimer, F., M.D.....Fourth and Sycamore Streets, Cincinnati
Freeman, Rowland G., M.D.....211, West Fifty-seventh Street, New York
Gittings, J. C., M.D.....3492, Chestnut Street, Philadelphia
Graham, E. E., M.D.....1713, Spruce Street, Philadelphia
Griffith, J. P. Crozer, M.D.....1810, Spruce Street, Philadelphia
Hamill, S. McC., M.D.....1822, Spruce Street, Philadelphia
Hand, Alfred, Jr., M.D.....1724, Pine Street, Philadelphia
Haynes, Royal S., M.D.....267, West Seventy-ninth Street, New York
Heiman, Henry, M.D.....56, West One Hundred and Twentieth Street, New York
Herrman, Charles, M.D.....250, West Eighty-eighth Street, New York
Holt, L. Emmett, M.D.....14, West Fifty-fifth Street, New York

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HOWLAND, JOHN, M.D.....49, East Fifty-third Street, New York
HUBER, F., M.D.....209, East Seventeenth Street, New York
JACOBI, A., M.D.....19, East Forty-seventh Street, New York
JENNINGS, CHARLES G., M.D.....457, Jefferson Avenue, Detroit
KERLEY, CHARLES G., M.D.....132, West Eighty-first Street, New York
KNOX, J. H. MASON, M.D.....804, Cathedral Street, Baltimore
KOPLIK, HENRY, M.D.....30, East Sixty-second Street, New York
LADD, MAYNARD, M.D.....295, Beacon Street, Boston
LA FÉTRA, LINNÆUS E., M.D.....113, East Sixty-first Street, New York
MACHELL, H. T., M D .....95, Bellevue Avenue, Toronto
MARTIN, C. F., M.D.....33, Durocher Street, Montreal
MEARA, FRANK S., M.D.....400, West End Avenue, New York
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MORSE, J. LOVETT, M.D.....70, Bay State Road, Boston
NICOLL, MATTHIAS, M.D.....124, East Sixtieth Street, New York
NORTHRUP, WILLIAM P., M.D.....57, East Seventy-ninth Street, New York
PISEK, GODFREY R., M.D.....36, East Sixty-second Street, New York
PUTNAM, CHARLES P., M.D.....63, Marlborough Street, Boston
RACHFORD, B. K., M.D.....323, Broadway, Cincinnati
ROTCH, THOMAS MORGAN, M.D.....197, Commonwealth Avenue, Boston
RUHRÄH, JOHN, M.D.....839, North Eutaw Street, Baltimore
SAUNDERS, E. W., M.D.....3003, Lafayette Avenue, St. Louis, Mo.
SHAW, HENRY L. K., M.D.....361, State Street, Albany
SNOW, IRVING M., M.D.....476, Franklin Street, Buffalo
SOUTHWORTH, THOMAS S., M.D.....807, Madison Avenue, New York
STARR, LOUIS, M.D.....1818, Rittenhouse Square, Philadelphia
TILESTON, WILDER, M.D.....308, Crown Street, New Haven
TOWNSEND, CHARLES W., M.D.....76, Marlborough Street, Boston
WENTWORTH, A. H., M.D.....352, Marlborough Street, Boston
WESTCOTT, THOMPSON S., M.D.....1833, Spruce Street, Philadelphia
WILLIAMS, HAROLD, M.D.....528, Beacon Street, Boston
WINTERS, J. E., M.D.....25, West Thirty-seventh Street, New York

---

HONORARY MEMBERS.

DR. JOHN THOMPSON.....Edinburgh, Scotland
DR. GEORGE F. STILL.....London, England
*DR. HENRY ASHBY.....Manchester, England
DR. O. HEUBNER.....Berlin, Germany
*DR. THEODOR ESCHERICH.....Vienna, Austria
DR. WILLIAM OSLER.....Oxford, England
DR. A. BAGINSKY.....Berlin, Germany
DR. V. HUTINEL.....Paris, France
DR. CHARLES RAUCHFUSS.....St. Petersburg, Russia

* Deceased.

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Deceased.

JOHN A. JEFFRIES, M.D.
Born, September 2, 1859,
Died, March 26, 1892.

THOMAS F. SHERMAN, M.D.
Born, March 17, 1856,
Died, September 26, 1893.

JOHN M. KEATING, M.D.
Born, April 20, 1852,
Died, November 17, 1893.

CHARLES WARRINGTON EARLE, M.D.
Born, 1845,
Died, November 19, 1893.

J. LEWIS SMITH, M.D.
Born, October 15, 1827,
Died, June 9, 1897.

JOSEPH O'DWYER, M.D.
Born, October 12, 1841,
Died, January 7, 1898.

JOHN HENRY FRUITNIGHT, M.D.
Born, November 9, 1851,
Died, December 18, 1900.

FREDERICK A. PACKARD, M.D.
Born, November 17, 1862,
Died, November 1, 1902.

WALTER S. CHRISTOPHER, M.D.
Born, 1859,
Died, March 2, 1905.

LEROY MILTON YALE, M.D.
Born, February 12, 1841,
Died, September 12, 1906.

JAMES PARK WEST, M.D.
Born, June 27, 1858,
Died, June 25, 1908.

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MINUTES OF THE TWENTY-SECOND ANNUAL MEETING OF THE AMERICAN PEDIATRIC SOCIETY.

Held at the New Willard Hotel, Washington, D. C., on May 3, 4 and 5, 1910.

FIRST SESSION—MAY 3D, 10 A.M.

The meeting was called to order by the president, Dr. David L. Edsall, of Philadelphia. The following members were present: Drs. Isaac A. Abt, Chicago; George N. Acker, Washington; Samuel S. Adams, Washington; A. D. Blackader, Montreal; David Bovaird, Jr., New York; Walter Lester Carr, New York; Henry Dwight Chapin, New York; F. S. Churchill, Chicago; D. M. Cowie, Ann Arbor, Mich.; John Dorning, New York; Charles Hunter Dunn, Boston; David L. Edsall, Philadelphia; F. Forchheimer, Cincinnati; Rowland G. Freeman, New York; E. E. Graham, Philadelphia; J. P. Crozer Griffith, Philadelphia; S. McC. Hamill, Philadelphia; Alfred Hand, Jr., Philadelphia; Henry Heiman, New York; L. Emmett Holt, New York; John Howland, New York; A. Jacobi, New York; Charles G. Kerley, New York; J. H. M. Knox, Baltimore; Henry Koplik, New York; Maynard Ladd, Boston; L. E. La Fétra, New York; Frank S. Meara, New York; J. Lovett Morse, Boston; Matthias Nicoll, New York; William P. Northrup, New York; B. K. Rachford, Cincinnati; Thomas Morgan Rotch, Boston; John Ruhräh, Baltimore; E. W. Saunders, St. Louis; Irving M. Snow, Buffalo; Thomas S. Southworth, New York; Louis Starr, Philadelphia; Charles W. Townsend, Boston; A. H. Wentworth, Boston.

There were present as guests: Drs. B. S. Veeder, of Philadelphia, and B. R. Hoobler, of New York.

The minutes of the twenty-first annual meeting were adopted as published in the ARCHIVES OF PEDIATRICS.

The following papers were read:—

1. President's Address: David L. Edsall, M.D., Philadelphia.

2. Drs. D. M. Cowie and William D. Lyons: "An Experimental Study of the Food Reactions in the Infant Stomach Compared with those *in Vitro*."

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3. Dr. A. H. Wentworth: "The Estimation of Chlorides in the Stomach Contents from Normal and from Atrophic Infants."

4 Dr. Henry Heiman: "A Study of the Gastric Contents and Motility in Breast-fed and Bottle-fed Infants."

5. Drs. Charles A. Fife and B. S. Veeder: "Studies of the Effect of Varying Qualities of Fat and of the Presence of Various Salts upon Digestion and Metabolism."

6. Dr. Henry Dwight Chapin: "The Nutrition of the Feeble Infant: (*a*) Motility of the Stomach; (*b*) Form and Quality of the Food Elements; (*c*) Results of a Trial of Finkelstein's Method; (*d*) Institutional *vs.* Fresh Air Treatment."

7. Dr. Maynard Ladd: "The Results of Substitute Feeding in Premature Infants."

8. Drs. John Howland and Robert A. Cooke: "The Effect of High Fat and Low Fat Diet upon the Nitrogenous Metabolism of Infants."

These papers were discussed by Drs. Wentworth, Meara, Chapin, Abt, Bernstein, Cowie, Heiman, Veeder and Howland.

9. Dr. J. P. Crozer Griffith: "Amyotonia Congenita."

This paper was discussed by Dr. Heiman.

10. Dr. John Lovett Morse: "A Study of the Value of Brudzinski's 'Neck Sign' and Contralateral Reflex in the Diagnosis of Meningitis in Infancy and Childhood."

This paper was discussed by Dr. Nicoll.

JOINT SESSION WITH THE AMERICAN ORTHOPEDIC ASSOCIATION—MAY 4TH, 10 A.M.

11. Dr. R. W. Lovett: "Infantile Poliomyelitis; Its Relation to the Community in Reference to Etiology and Prevention."

12. Dr. L. Emmett Holt: "The Clinical Aspects of Poliomyelitis; Types; Communicability; Mortality."

13. Dr. Simon Flexner: "Experimental Epidemic Poliomyelitis and its Relation to Poliomyelitis in Human Beings."

14. Dr. Irving M. Snow: "Paralysis of the Neck and Diaphragm in Poliomyelitis."

15. Dr. Israel Strauss: "The Pathology of Poliomyelitis."

16. Prof. D. Fritz Lange: "Treatment of the Amelioration

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of Permanent or Partial Paralyses by Silk Tendons and Silk Ligaments."

These papers were discussed by Drs. Sachs and Orr.

THIRD SESSION—MAY 4TH, 3 P.M.

17. Dr. Thomas Morgan Rotch: "A Comparison in Boys and Girls of Weight, Height and Epiphyseal Development."

18. Dr. Charles Hunter Dunn: "The Cytodiagnosis of Tuberculous Meningitis and the Possibility of Recovery."

This paper was discussed by Drs. Northrup, Meara, Hamill, Griffith, Churchill, Abt, Dunn, Adams. Dr. Dunn closed the discussion.

19. Dr. S. McC. Hamill: "General Considerations Regarding the Effects of Vaccines."

20. Drs. John Howland and B. R. Hoobler: "The Use of Bacterial Vaccines in Children's Diseases."

These papers were discussed by Drs. Churchill, Koplik, Saunders and Howland.

21. Dr. A. D. Blackader: "Thymic Asthma."

22. Dr. F. Forchheimer: "The First Sound of the Heart in Children."

This paper was discussed by Drs. Northrup, Hamill, Churchill and Forchheimer.

23. Drs. J. P. Crozer Griffith and L. E. La Fétra opened the discussion on "The Value and Limitations of the Employment of Cold Air in the Treatment of Acute Respiratory Conditions."

The discussion was continued by Drs. Northrup, Graham, Howland, Blackader, Southworth and Abt. Dr. Griffith closed the discussion.

24. Dr. W. P. Northrup: "Retained Intubation Tube: Abductor Paralysis (Diphtheria): Recovery."

FOURTH SESSION—MAY 5TH, 10 A.M.

25. Dr. F. S. Churchill: "The Bacteriology of the Blood in Early Life."

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This paper was discussed by Drs. Holt, Koplik, Abt, Bovaird, Edsall and Churchill.

26. Dr. H. Koplik: "The Chronic Rheumatoid Affections of Childhood."

This paper was discussed by Drs. Abt and Koplik.

27. Dr. David Bovaird, Jr.: "Acute Pericarditis in Children."

This paper was discussed by Drs. Churchill, Koplik, Abt and Bovaird.

28. Dr. D. M. Cowie: "Chylous Ascites in Infants."

This paper was discussed by Drs. Bovaird, Adams and Cowie.

29. Dr. J. H. M. Knox: "The Importance of a More Complete Registration of Births in the United States."

The following papers were read by title:—

"Obituaries on A. Steffan and Alois Monti," by Dr. A. Jacobi.

"Clinical Phenomena Associated with the Late Development of the Pyramidal Tracts," by Dr. B. K. Rachford.

"A Study of Eighty Cases of Empyema in Children," by Dr. Francis Huber.

"A Case of Typhoid Mania in a Child," by Dr. Allen Baines.

EXECUTIVE SESSION—MAY 5TH, 10 A.M.

The report of the Council was read by Dr. L. Emmett Holt, its recommendations being adopted as follows:—

As officers for the ensuing year:—

President, Dr. Henry D. Chapin, New York; Vice-President, Dr. J. H. M. Knox, Baltimore; Secretary, Dr. Samuel S. Adams, Washington; Treasurer, Dr. Charles Hunter Dunn, Boston; Recorder and Editor, Dr. L. E. La Fétra, New York; as a new member of the Council, Dr. Isaac A. Abt, Chicago.

Recommended for membership, Dr. H. L. Coit, Newark, N. J.; Dr. G. R. Pisek, New York; Dr. R. S. Haynes, New York; Dr. J. C. Gittings, Philadelphia; Dr. Charles Herrman, New York; Dr. Wilder Tileston, Boston.

The resignation of Dr. Booker was accepted.

The Council recommended an assessment of $15 for the coming year.

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The Council recommended the following resolution (read by Dr. Holt):—

Resolved: That the members of the American Pediatric Society heartily endorse the plan proposed for a new journal of diseases of children and if it is established pledge their cordial support.

The Council recommended that two committees be appointed, to be known as, first, Committee on Child Hygiene, to which all matters referring to that subject shall be referred; second, Committee on Infant Mortality; these committees to be appointed by the President and to coöperate with outside organizations whenever such coöperation is called for.

The President appointed as the Committee on Child Hygiene Drs. Jennings, Rotch and Hamill; as the Committee on Infant Mortality Drs. Holt, Churchill and Knox.

The committee recommended the endorsement of the work of the Russell-Sage Foundation in regard to the matter of Child Hygiene.

Dr. Adams moved that the President be instructed to cast an affirmative ballot for the officers as nominated by the committee; seconded, carried, and the ballot so cast by Dr. Edsall.

On motion duly seconded, the Secretary was instructed to cast the ballot for those recommended for membership, which was accordingly done.

Dr. Northrup moved that the Society adopt the resolution of the Council as to the establishment of a new journal; motion seconded and carried.

Dr. Northrup then moved that the other recommendations of the Council be adopted; motion seconded and carried.

The Secretary read a letter from the American Orthopedic Society containing a resolution relative to making poliomyelitis a notifiable disease, and moved its adoption; seconded and carried.

The time and place for the next annual meeting were set for Lake Mohonk, May 31, June 1 and 2, 1911.

SAMUEL S. ADAMS, M.D.,
Secretary.

L. E. LA FÉTRA, M.D.,
Recorder.

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ANNUAL ADDRESS OF THE PRESIDENT, AMERICAN PEDIATRIC SOCIETY.

BY DAVID L. EDSALL, M.D.,
Philadelphia, Pa.

The honor of serving for a year as the president of a group of men who are the highest representatives of their type of work in this country imposes upon one, as I conceive of it, the duty not merely of giving expression to complacent satisfaction, but also of devoting serious thought to the activities that may be open to the members of the Society as individuals or as a group by means of which they may still further advance the honorable standing of the work that is their chief concern in life. I am in a somewhat dangerous position in approaching this duty, for since my own work has come to be more largely outside the field of pediatrics than in it I may easily find myself presenting much the same appearance to you, the masters of pediatrics, as does the occasional maiden lady of philanthropic tendencies who would instruct mothers in suitable methods of training children. This is the more likely to be the case inasmuch as the attempt to urge new activities upon you involves not only the giving of unsought advice to those who are my superiors, but also it carries with it some suggestion of criticism in that it presupposes that I consider those things that have been done to be not yet sufficient. I recognize fully, however, that the things that I have to say cannot be considered exactly as advice, inasmuch as they are not my own thoughts alone, but in more or less the same form are uppermost in the minds of many others among you; and as to any flavor of criticism, this is due not to any lack of appreciation of what has been done, but to my feeling that in spite of all that has been done, proper dignity and regard are not yet generally accorded in this country to a subject which, it appears to me, is unquestionably the broadest and most complex and the most important division of general medicine, and proper standing will not be generally given to pediatrics in this country until better things are demanded for it by concerted action, and until also better things are in certain ways demanded of it by a large group of those who are the recognized leaders.

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The dignity of any of the intellectual pursuits and the standing which it has in comparison with other things related to it are determined chiefly by two things. First of all, I think, by the opportunities offered those who teach the subject and by the manner in which these opportunities are made use of; and, second, by the extent to which those who follow that pursuit engage in matters that favorably affect the welfare not only of those individuals with whom they come in immediate contact, but rather of the public at large. There are, it appears to me, ways in which greater usefulness could be secured for pediatrics in both of these directions, and I believe that this society could rapidly, by the strength of carefully planned concerted action, greatly benefit the position of the work which it represents. It may appear to many of you somewhat out of place for me in addressing this society to devote most of my time to the question of the teaching of pediatrics, but I think an apology for doing so is hardly necessary, partly because most of you are teachers, but also for a much broader reason than this; that is, it is undeniable that while teachers are far from constituting the only persons who have a determining influence upon the progress of their subject, they do have a most serious responsibility, not only because each of them has charge of a limited group of students, but much more because the methods they pursue chiefly determine the standards that are demanded in that subject, and even more important than this the teacher and his methods largely determine the quality of the future exponents of his subject, not merely through the actual information that he disseminates among students, but through his directly attracting a better or worse group of men into that subject as their life work. The appearance of attractiveness of any subject to those who have not yet chosen their own special careers is most largely dependent upon the inherent interest in the subject itself and upon the breadth of view and enthusiasm of the teacher, but it is also, and often equally largely, dependent upon the halo which is given to the subject by the facilities which are provided for the teacher. It would be quite unsuitable and unnecessary for me to enter into a discussion of the worth of individuals who have charge of the teaching of pediatrics in this country. I may dismiss this point by saying that among their number are men who have in certain ways no superiors in any part of the world. Concerning the provision made for their teach-

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ing, however, there are points which may justly be commented upon and not wholly favorably. I recently had a long discussion regarding medical education with one of the ablest educators in this country, and he told me most forcibly that an extended inspection of medical schools had convinced him that, considering its importance, pediatrics is more inadequately provided for in most medical schools than any other important subject; and he frankly said that, considering the importance inherent in this subject, the conditions surrounding it are, in most places, shameful. He had investigated almost solely the best schools in this country. On the whole, I think we must agree with him in large part. I would hasten to say that his criticism was of the general group of schools that he had seen, not of all the individual schools, and it does not apply in a number of instances where the system of teaching is relatively excellent, but at the same time I would say that nowhere is the provision made for the subject as good, even relatively, as that which is made for general medicine, general surgery and obstetrics in a number of schools where these subjects are particularly well cared for. In fact, I may fairly say that a more directly comparable branch of work, neurology, has much more actual standing and recognition in the general group of our best medical schools than has pediatrics; yet I am personally convinced that the problems of infancy and early childhood are much more distinct from general medicine than are the problems of neurology, and consequently they more properly demand generous provision for instruction in them. They are at the present stage of our knowledge surely very much more important practically to the student.

An extremely striking, and, I believe, direct, result of the existing academic state of pediatrics is that not only does the individual student receive in most places very inadequate training in the subject; even more significant than this is the fact that fewer able young men than might properly be expected to do so go into pediatrics as contrasted with the number of such men that go into general medicine, and the number is less even than of those who go into neurology; for at the present day we may certainly say that there are in this country at least twice the number of men of recognized distinction in neurology that there are in pediatrics. The explanation for this is, I think, a reasonably patent one, and while the responsibility may properly be thrown

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in part upon the shoulders of the administrators of medical schools, it cannot be put wholly upon them and must be shared by pediatrists themselves.

It is a common, and, on the whole, a seemingly just, statement that the clinical branches have in this country in the past generation by no means kept pace with the medical sciences in their methods of teaching and investigation. I would especially point out here that in mentioning research as I have just done, I do it purposely, but not at all with the idea of pleading at the moment for research. I speak of it with the view of pleading for better general conditions in teaching, for it is, I believe, a well recognized fact among educators that in most instances, unless a man does some kind of research more or less continuously, he does not make or, at any rate, does not long remain, a sound teacher, simply because the teacher must, in order to make the student search for knowledge, search diligently and continuously himself. Therefore, aside from the usefulness of knowledge gained through research, the kind of research work that is produced in any particular department is commonly accepted, and rightly so, as the best individual criterion of the degree of progressiveness that the teaching in that department will exhibit. Now during the past generation the medical sciences have been transformed from subjects that served chiefly to give those who taught them dignity and high position in closely related forms of practice, into subjects that of themselves furnish chosen and enviable careers to many of the best men in the profession. An inevitable and happy result of this has been that as they have assumed greater dignity and seriousness they have demanded and secured greater facilities and opportunities for teaching and research, and have in both these ways advanced with tremendous rapidity.

With the clinical branches no such fundamental change in point of view has occurred and none is possible; for continuous touch with practical matters is essential in the teaching of things that are necessarily practical, and therefore the teaching of clinical work cannot be wholly separated from practice. It has been impossible consequently to start wholly fresh in these subjects upon an altogether new basis, and hence whatever change there has been in them has been in details rather than in general plan. Hence they still retain some of the casualness in teaching and research that the medical sciences cast off when they became

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purely academic subjects. Nevertheless there have been very significant forward steps in these branches and the occasional criticism of them that they have shown little, if any, progress is not by any means as well justified as it seems upon superficial consideration. I sincerely believe, however, that such a criticism is better justified when directed at pediatrics than when any other of the most important subjects in medicine is aimed at.

The chief essentials for making well trained men and for attracting first-class men into a subject as their own work are that the men shall do most of the work themselves and not merely be told about it; that they shall be made precise and critical in their methods, and that the craving for a sound understanding of the reasons for things be satisfied by having them clearly comprehend the relation between the fundamentals and the practical things. In all these ways general medicine and general surgery have been quite transformed within two decades in a large group of our best medical schools, and now, instead of the instruction being as it was earlier, almost wholly didactic and at best given in the form of clinical lectures to large classes, students do most of their work in wards and dispensaries, either in small groups or, still better, as individuals, acting practically as temporary assistant internes. Precision and a critical point of view are given them through the fact that purely observational methods, which always have an indeterminable amount of error associated with them, are supplemented by the employment of laboratory methods and instruments of precision, which not only give added information, but have the invaluable element of demanding accuracy and precision in their use and thus training into more precise habits. The craving for a fundamental understanding of facts that are rather baldly stated in text books is, in some places at least, being vigorously met by coöperative teaching between clinical and anatomical, physiological, pathological and other departments, or by much greater attention to these matters on the part of the clinical teacher, so that in either case the important bearings of the fundamentals upon clinical matters are brought prominently forth in immediate relation with the clinical teaching. In fact, a fair knowledge of physiology and pathology have become almost as important a part of a clinical teacher's training as is clinical experience itself, and that this is well recognized grows increasingly evident when one notes the character of a consider-

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able group of appointments to important chairs in recent years. What I have said in regard to general medicine and general surgery is, to a large extent, true also of neurology, and to some extent of certain of the other specialties. There has likewise been movement in this regard in obstetrics and gynecology, though perhaps to a less degree than should have been the case. When we consider what has occurred in pediatrics I think we must admit that the changes have been much slighter and less distinctive. There has been decidedly less opportunity given the student to work himself upon cases, and in many of the best schools he still sees children solely, or almost solely, through the eyes of the instructor. This is due to the fact that while the opportunities for actual work in wards and for bedside instruction in small classes have been growing everywhere in medicine and surgery, there are scarcely anywhere pediatric wards where students are freely permitted to do things themselves. The critical difference then is that while in medicine the student not only may, but in the best departments must, get extensive training in working out for himself the history, the physical examination, the examination of secretions, etc., in a series of cases, and often must, under the control of the instructor, plan out and follow the treatment, in pediatrics he usually does but a fraction of this; and as a necessary result cases are less frequently seen as problems to be worked out by the student himself than as problems which the instructor both points out to him and solves for him. Naturally this is less attractive to the best men. Healthy-minded persons enjoy eating more than they do being fed. Guidance by instructors is, of course, essential, but it must be guidance in the methods of attacking problems rather than in the actual solving of these problems, especially when the students reach their final year. Furthermore, unless they do the work themselves the lack of opportunity of bringing for themselves all the methods of precision to bear upon each case leads students into less exact methods and into less respect for the subject that they are dealing with. Again, as I said, I believe the serious-minded student takes a deep interest in any subject only when he has clear conceptions of its relations to those things on which it is based. This is to my mind the most important side of the matter that I am discussing, though not usually dwelt upon. It is, it seems to me, the crux of the whole situation in so far as the character and number of the men going

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into pediatrics is concerned. So far as I can see, the propriety of studying pediatrics as a special subject rather than as a mere part of general medicine is based essentially upon the fact that the infant and young child differ from adults enormously in degree and almost equally in kind, in susceptibility to infections, and especially in liability to derangements of nutrition. This constitutes a really profound difference, it appears to me, and gives much greater justification for specialization than any that is based upon the different organs of the body, for it demands of those who would enter into pediatrics in a broad spirit very wide and subtle knowledge of the two types of work that are now, and probably will be for years to come, the most progressive and most productive lines of work in all medicine, but that are at the same time the most complex and difficult. I mean, on the one hand, infection, immunity and hygiene; on the other, the physiology and pathology of nutrition. In spite of the fact that these subjects are simply teeming with points that are of the most intense interest in acquiring sound and broad methods of practice, there are certainly scarcely any places where an attempt is made to bring out systematically and thoroughly their relation to clinical pediatrics. So far as these subjects are at all broadly dwelt upon by clinical teachers, it is almost entirely in courses on general medicine not in pediatrics, though they are of even more intense interest practically in the latter. It is no wonder that in the eyes of many of the best students pediatrics becomes the tail to the general medical kite, when the things most intimately related to it are largely neglected in direct relation to it, but are taught in practical relation to other clinical subjects. As I have intimated, however, I think that we may more justly compare the conditions surrounding pediatrics to those in neurology rather than to those in general medicine, and, as I have said, to the disadvantage of pediatrics. But the same explanation of the different status of the two may be used here: In every place where neurology occupies a conspicuous position in the curriculum and is highly regarded by students it will be found that the fundamentals are most intimately related to the clinical teaching, and there are special courses, usually directly associated with the clinical teaching, for broadening the student in his knowledge of the anatomy, physiology and pathology of the nervous system.

The manner in which this disadvantageous state of things

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can be overcome is, I think, indicated by what I have just said. Students should be taught in the pediatric department, or by the pediatric department in association with other departments—and not simply in some department entirely separate from pediatrics—the important direct practical bearings of the study of the physiology and pathology of nutrition, of infection and immunity, and of all the fascinating things that fall under the name hygiene.

The overcoming of the difficulties in regard to the studying of cases can be accomplished only through demanding that the clinical work in pediatrics shall be put on the same basis as that which is being struggled for, and increasingly secured in medicine and surgery; that is to say, there shall be pediatric hospitals, or at least wards of really adequate size where the department shall be absolutely at liberty not only to show cases, but to put the students at work in whatever way it is believed will lead to the best training for them. This is an old story in medicine now, and no teacher in clinical medicine is content with anything less whether he has obtained it or not; especially is he not content with a rotating service where the system changes with each change in the service, where he is thoroughly familiar with the cases only during his own term of service, where any investigation must be wholly fragmentary and interrupted and where everything is more or less dependent from day to day upon the goodwill of his colleagues and the hospital managers. Pediatrists have shown very much less evident activity in attempting to secure proper conditions of this kind and very much less apparent appreciation of the importance of such conditions than have those who are engaged in general medicine, surgery and obstetrics, and yet such conditions are, I had almost said, more important than in the other subjects mentioned, for little details that one becomes familiar with only by direct contact with them, and not merely by hearing of them, are determining factors in successful diagnosis and treatment with children almost more than with adults. Nowhere in this country are the conditions as good yet as they are in a number of places in the other subjects mentioned. In two schools there will soon be departments with model systems even better than the best that now exist, where the head will have entire authority in a good hospital, will have the academic dignity and the salary that go with high ability and wide training, and will have the power to plan his teaching in whatever way ap-

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pears most efficient without regard to the fancies of the hospital managers; and where in return for these privileges it will be expected of him that he shall keep himself thoroughly trained, not only in pediatrics itself, but in the lines of work that it most intimately touches upon, and that he shall not only disseminate knowledge, but contribute to it, and train others to do the same. Certain of our most distinguished members have previously established eras in pediatrics in America, and one member, who is still stimulating us to higher ideals and better methods, is the veritable parent of the subject in this country. I anticipate that these new clinics will inaugurate another new era partly through the standards that they will set and that others must follow, but even more largely through the considerable group of highly trained men that the facilities provided in them will almost certainly lead into pediatrics as their life work.

Until conditions such as those that I have been discussing are generally recognized as the standard that is being striven for, I do not believe pediatrics will assume the station that it deserves in comparison with other divisions of medicine. Certainly dependence upon hospitals that are not thoroughly controlled through either ownership by the school or firm affiliation with it is an anomaly that is almost as damaging to the best ideals in medical education at the present stage of things as was the money-getting spirit of the teaching in the days of the old proprietary schools. It appears to me that this society could wisely take upon itself a serious consideration of the manner in which teaching and investigation in this branch may be benefited, and then by both individual and concerted action further whatever appears to be wise. It is beyond question that acting as a group and with the power that comes through the name of the American Pediatric Society much could be rapidly accomplished. At this very time I know that a committee is preparing a report to one of our sister societies on the question of securing freer use of clinical material by students and better teaching facilities in other ways in the subject which that society represents. An authoritative report of this kind which will be recognized as representing not the ambitious desires of one man, but the earnest opinion of a commanding group of men, will have much force in bettering things if wisely used; indeed, I know of one or two schools that, knowing what this report recommends and desiring not to be found

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wanting when it appears, have already anticipated it and have advanced their standards to the point which it will demand.

In the earlier part of my address I spoke of the relation of men to the public at large as one of the main ways of being useful and one of the chief ways by which their usefulness is judged. There are a variety of ways in which this society could, I believe, act as the center for the dissemination of education, and particularly as the means through which knowledge could be acquired by us and by others interested in the subject in a considerable number of things which bear intimately upon the subject of pediatrics and which are of great importance to the public at large, but in which reliable information is difficult for every one of us to secure. Some of these were discussed to a slight extent at last year's meeting. The conditions in children's schools could probably be led into a much better state than they are now were there some sort of conference between important educators in these schools and representative pediatrists such as might be furnished by a committee of this society. Industrial conditions among children have deeply interested me in connection with industrial conditions in general, and there is no question that they need study by persons of calm and judicial temperament so that we may be informed upon the actual state of affairs by those whose opinions would be more reliable than those of perhaps over-enthusiastic philanthropists. I do not agree with the view expressed last year that such questions are outside the realm of research and are rather matters of mere medico-legal interest. The more I have studied occupational disorders the more fruitful a field of research I have found them. There are a number of other important questions relating to other parts of hygiene in which I believe this society could take a useful part and a part that would in no way interfere with its other activities; and it could thereby cover subjects which are at present not particularly cared for by any other group of qualified persons, and concerning some of which it is now well-nigh impossible to get any satisfactory information or to offer reliable advice. Such matters I cannot take up *in extenso*. as I have already consumed an undue amount of time. I should be very glad, however, to see members of the society, or the society as a whole, consider the relation of the society to such matters, and either proceed to take some active part in them or condemn any such plan definitely.

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A STUDY OF THE STOMACH CONTENTS AND MOTILITY IN BREAST- AND BOTTLE-FED INFANTS.

BY HENRY HEIMAN, M.D.

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In contrast to the fairly uniform results derived from the study of the gastric functions of the adult is the marked lack of uniformity of the data obtained from the investigation of the digestive functions of the stomach of infants. This striking fact is not attributable to lack of careful investigations, but in part to the employment by successive investigators of a number of different chemical methods of varying degree of accuracy; in part to the inability of employing certain valuable methods of study which, though harmless for the adult, are inapplicable for the delicate organism of the infant, but in greater part to the well-known physiologic variations of the digestive functions at this period of life.

Although Kussmaul invented the stomach tube in 1867, it was not until 1880 that this invaluable instrument was first used in infants and children. In that year Epstein demonstrated the entire harmlessness of the introduction of the tube into the stomach of even very young babies, and called attention to the value of its application in the treatment of gastric diseases of infancy and childhood. Though he did not primarily employ the stomach tube for diagnostic purposes, he did incidentally find that the stomach in very young normal infants was empty after one to one and one-half hours. The first use of the stomach tube for scientific investigation was made in 1887 by Raudnitz, who determined the presence of rennet in the gastric contents of three infants from two to six months of age. In four other infants in the first week of life he was unable to find rennet. Since 1887 almost every year has brought forth important contributions to the subject of gastric digestion in infancy. The literature to date is so voluminous that I shall of necessity mention only the most important papers. I shall refer in some detail to the papers of

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Leo in 1888, Von Puteren 1889, Heubner 1890, Pipping 1891, Pfannenstill 1892, Czerny 1893, Von Hecker 1900, Meyer 1903, Hamburger and Sperk 1905, Langstein 1905, Sedgwick 1906, Rosenstern 1908, and Reeve-Ramsey 1908.

The first important and classic paper was that of Leo, who examined 30 normal infants from the age of two hours to twelve months, and 104 infants suffering from various gastrointestinal diseases. In the normal breast-fed infants he found that the stomach emptied itself in about one and one-half to two hours, and during the first week of life in one hour. In artificially-fed infants and in the older children of the series the food remained longer in the stomach. The total acidity was higher in the artificially-fed infants and its percentage increased with the duration of gastric digestion, but even in these cases no free HCl was found until after one hour. This author first called attention to the importance of the "combining power" of milk with HCl. This fact accounts for the non-appearance of free HCl until after complete saturation of the milk. He determined the presence of pepsin in all the cases and found in contrast to that of Raudnitz that rennet was present even in the newborn. His finding of a diminished number of bacteria in the gastric contents, as contrasted with the number originally in the food before ingestion, led him to attribute great importance to the antifermentative (bactericidal) properties of the gastric juice.

This bactericidal action of the gastric juice could not be confirmed by Von Puteren, who examined 248 infants, all below six weeks of age. This investigator not infrequently found food in the stomach after two hours, at which time free HCl was almost always present. The total acidity in his cases varied from .03 per cent. after ten minutes to .08 per cent. after ninety minutes, which was the maximum. During the first four weeks of life he was unable to demonstrate the presence of rennet.

Heubner, in 1890, reported on about 40 infants, the ages varying from two weeks to fifteen months. He made special studies on the presence of lactic and volatile acids and found lactic acid present in 4 breast-fed infants and in 9 infants fed on cow's milk. Volatile acids were found by him in 5 out of the 26 artificially-fed babies.

Pipping, who examined 29 normal infants under three months of age, found that the stomach emptied itself in about two hours. He confirmed Heubner's results in demonstrating lactic acid more

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frequently in artificially-fed infants. While butyric acid was never present in the breast-fed, in the infants fed on cow's milk this acid was constantly present.

Czerny again drew attention to the "combining power" of milk with HCl. He found that cow's milk takes up more HCl than does human milk. In order to retain the bactericidal properties of the gastric juice which is due to free HCl, he urged the advisability of longer intervals between feedings, especially when cow's milk is used. This author also found that whereas the stomach of a young infant is empty after an interval of two hours, when several feedings are given in rapid succession, considerable remains of the last meal may be found even after three or four hours. This fact in his opinion also speaks for the advisability of lengthening the feeding intervals.

Pfannenstill first employed the salol test for the determination of the time required for food to pass into the intestines. He administered salol and examined the urine, obtained by means of a catheter kept in the bladder during the entire period of examination, at intervals of five minutes. In 60 healthy children he found that the ferric chlorid reaction appeared in the urine from forty to sixty minutes after ingestion of food mixed with salol.

Von Hecker, employing the same method as Pfannenstill, found that the time of appearance of the ferric chlorid reaction varied with the age of the child. The younger the child the earlier the reaction appeared.

Meyer discarded the use of milk for most of his investigations and employed barley water and occasionally sterilized water or physiological salt solution as a test meal. He claims that with these media organic acids do not appear, and that the presence of HCl in its free and combined state is thus more readily determined. With this test meal he found in 17 artificially-fed children the stomach empty after one to one and one-third hours. While in most cases the total acidity increased with the duration of gastric digestion, this was by no means constant. He attributes the occurrence of these variations to at present unknown nervous influences.

Hamburger and Sperk made an extensive investigation on 63 normal breast-fed infants. They found no free HCl until after one hour and no lactic acid at any time. They attribute considerable theoretical and some practical importance to the so-called HCl deficiency, which is determined by adding a decinormal HCl

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solution to the gastric contents to the point where free acid becomes demonstrable. They made the interesting observation that the "combining power" of the gastric contents (as measured by the total acidity plus HCl deficiency) was greater than that of an equal amount of milk, before it was taken into the stomach. While the explanation for this unexpected phenomenon is not entirely clear, the authors think that it is probably due to the secretion into the stomach of acid salts, weak organic and fatty acids.

Langstein made careful chemical investigations on the protein digestion in infants and came to the conclusion that peptones were the result of the action of pepsin exclusively, and that rennet played no part in this process.

Sedgwick found that a fat splitting ferment occurs in the infant's stomach as early as the second week of life, and that a part of the acidity of the gastric contents may be due to the action of this ferment.

Rosenstern employed the ricin method for the quantitative estimation of pepsin in the gastric juice. His results showed that the quantity of pepsin secreted increases during the first four months of life and that after this period it remains constant, and that artificially-fed infants secrete more pepsin than breast-fed infants of the same age.

Reeve-Ramsey, employing the edestin method for his investigation, found that pepsin is always present in the normal breast-fed infant, and that this ferment in the gastric juice can split up protein into peptones without the addition of acids; and that hydrochloric and lactic acid and pepsin are not necessarily always present at the same time. From a survey of the literature mentioned it can readily be seen what radical differences of opinion exist among investigators as regards practically all the important functions of the infant stomach.

The present investigation was not undertaken with the idea of solving the many mooted questions in this field of the physiology of the infant, but for the purpose of adding another series of data to American literature, which up to the present time has not been very prolific in investigations on the gastric digestion of infants.

The cases studied were divided into two groups. The first group included 20 normal infants from three to seven days old, born at the New York Lying-in Hospital. The second group comprised 13 artificially-fed infants from six weeks to seven

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months of age, all convalescent from various acute diseases, for which they were admitted to the children's service of Dr. Koplik, at the Mount Sinai Hospital of New York City. In all these cases the temperature had been normal for at least a week and the general condition good, so that for our purposes they may all be considered to have been at the time practically normal infants.

The method of procedure with the newborn infants was as follows: Where practicable, that is in the majority of the cases, the baby was weighed before and after nursing, so that the quantity of milk taken could be accurately determined. At the end of one-half and one hour respectively, on alternate days, the stomach contents were removed by means of a No. 18 (Fr.) Nélaton catheter. The introduction was readily performed while the child lay on its back and was well borne by all the infants; some of them continuing to sleep during the greater part of the procedure. In most of the cases the gastric contents were more readily obtained when the infants began to cry or gag. At the end of the procedure an aspirating bulb was attached to the stomach tube, but in no case could more contents be thus obtained. No water was introduced into the stomach for the purpose of ascertaining that it was completely empty, nor was preliminary gastric lavage undertaken. Change of position, or even inversion, had no marked influence on the quantity of contents obtained from the stomach. The chemical methods used were the same as those for the older bottle-fed infants and will be described later.

In the second group four examinations were made for each individual case, namely, at the end of one-half, one, two and three hours respectively, on alternate days. In a few cases the stomach tube was introduced at the end of four hours, but in only one were contents obtainable. No greater difficulty was encountered with the older infants than with the newborn in the removal of the gastric contents. No preliminary gastric lavage was undertaken, as it was found impracticable to do so. The contents were received in a glass jar, then measured in a graduate. If the volume was less than 20 c.c. an equal amount of distilled water was added and the calculations made accordingly. After ascertaining the volume the contents were poured on a filter. The filtrate was then used for the following determination:—

Total Acidity.—5 c.c. were titrated with $^{N}/_{10}$ NaOH solution, phenolphthalein being the indicator.

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Free HCl was determined by Congo paper and phloroglucin-vanillin. When the reaction was positive 5 c.c. were then titrated with Töpfer's reagent.

Pepsin.—5 c.c. of stomach contents were added to a Mett's tube 1 c.c. long, then placed in a thermostat over night and the amount of digestion noted.

Rennet.—5 c.c. of fresh milk were added to 5 c.c. of gastric contents, then placed in a thermostat and observed at intervals of twenty minutes for one hour for curd formation.

Lactic Acid.—To the remaining amount of stomach contents were added 10 drops of H_2SO_4, then put into a separator and shaken with equal volume of ether; the water layer was then run off and the ethereal solution filtered; to this filtrate was then added a very dilute solution of $Fe_2 Cl_6$ and shaken in a flask. In presence of lactic acid the H_2O solution turns canary yellow.

A microscopic examination of the sediment of the gastric contents before filtration was made in a number of cases, and practically nothing but fat globules and starch granules were seen.

I shall now proceed to a discussion of the principal finding in each of the two groups.

In the first group there were 20 newborn infants and 36 specimens of gastric contents were examined. Of the 20 infants 1 was three days old, 1 four days, 2 six days, 1 seven days, and 15 five days old. The quantity of milk consumed at each feeding, as measured by the weight of the infant before and after nursing, varied from 30 to 200 grams, the average being about 100 grams, a quantity which is considerably above the figure which has been assumed to be normal at this time of life. The quantities recovered at the end of one-half hour varied from nothing to 60 c.c., the average quantity being 25 c.c. There was apparently no relationship between the quantities consumed and the quantities recovered. At the end of one hour the figures varied from 4 c.c. to 45 c.c., the average for the 16 cases examined being 24.3 c.c. In several instances, as can be readily seen from the table, the amount obtained at the end of one hour was greater than that at the end of one-half hour (in the same infant). In 5 additional cases, in which the stomach tube was introduced from two hours and fifteen minutes to two hours and thirty minutes after nursing, about ½ to 3 c.c. of fluid containing a few curds was removed.

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The *total acidity* at the end of one-half hour varied from 10 to 112, the average being 31.2.

In the 16 cases in which the contents were removed at the end of one hour the total acidity varied from 12 to 60, the average being 29.2. These findings are not in accord with those reported by most observers, who found a progressive increase in acidity during the course of gastric digestion.

Free HCl was found in none of the cases.

Pepsin.—Without the addition of HCl no peptic action (Mett's tube) could be determined in any of the cases. When one drop of a 2 per cent. HCl solution was added to the gastric contents the presence of pepsin could be demonstrated in all but 4, and even in these pepsin was present in the contents removed at the end of one hour. In general, the peptic action was more marked in the one hour than in the one-half hour contents.

Rennet.—In the one-half hour contents there were 7 specimens out of 20, or 35 per cent., in which no rennet was found. In the one hour cases there were 8 out of 16, or 50 per cent. negative specimens. In this connection I wish to call attention to the fact that the latest researches of Pawlow and Jacoby seem to indicate that rennet is not a special ferment, but that the curdling of milk is due to the action of all proteolytic ferments, which split up caseinogen into a substance which combines with calcium salts to form the curd.

Lactic acid was present in 10 specimens. It was marked in 3 cases, moderate in 2, and faint in 5 cases. In general, it was more often found in the one hour than in the one-half hour contents.

In the *second group* (the bottle-fed babies) there were 13 cases and 39 specimens of gastric contents were examined. The ages varied from six weeks to eleven months. The quantities recovered after one-half hour varied from 4 to 146 c.c.

At the end of one hour the stomach was empty in 1 case (six months old; quantity of feeding, 90 c.c.). The maximum quantity of contents obtained was 103 c.c. (this child being five months old; quantity of feeding, 180 c.c.). At the end of two hours the stomach was empty in 4 cases and the maximum quantity recovered was 60 c.c. (in 2 children, one seven months and the other eleven months old; quantity of feeding in each case, 240 c.c.). At the end of three hours the stomach was empty in

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5 cases and the maximum quantity of contents was 35 c.c. (in a child eleven months old, who took 240 c.c. at a feeding). On a subsequent examination the same child yielded 40 c.c. at the end of four hours. In 7 other cases nothing was obtained at the end of four hours.

Total Acidity.—In the one-half hour specimens it varied from 10 to 36, the average being 22.3. In the one hour specimens it varied from 3.75 to 41.4, the average being 28.7. In the two hour specimens it varied from 4 to 70, the average being 44. In the three hour specimens there were three examinations. One case showed an acidity of 8, a second specimen an acidity of 54, and a third an acidity of 75.

Free HCl was present in but 2 cases. In one case it was 12.4 after two hours, in the other case it was 4 after one-half hour, 31 after two hours and 38 after three hours.

Pepsin was present in 6 cases; in these it was not found necessary to add HCl to develop its proteolytic action.

Rennet was present in all but 2 cases.

Lactic acid was present in 3 cases; in 1 at the end of one-half hour, and in 2 at the end of one hour.

CONCLUSIONS.

Fully recognizing the fact that no important conclusion should be drawn from the examination of but a small number of cases, yet I may be permitted to call attention to a few interesting results which are brought out by the present study.

The Newborn Infant.

(1) The large amount of milk taken at one nursing by many of the newborn infants.

(2) The fact that practically the same quantity of gastric contents was obtained at the end of one hour as at the end of one-half hour, and that at the end of two to two and one-half hours ½ to 3 c.c. could still be recovered.

(3) The fact that the total acidity was practically the same at the end of one hour as at the end of one-half hour.

(4) The absence of free HCl in all the cases.

(5) The demonstration of pepsin on addition of HCl in all of the one hour specimens and in all but 4 of the one-half hour specimens.

(6) The presence of rennet in two-thirds of the cases.

(7) The presence of lactic acid in one-half of the cases.

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Bottle-fed Infants.

(1) The stomach was found empty at the end of two hours in 4 out of 13 cases, and in 5 out of 9 cases at the end of three hours, and in 5 out of 6 cases at the end of four hours.

(2) In contrast to the breast-fed infants, in this group there was a noticeable increase in the total acidity from one-half to three hours in most of the cases.

(3) The presence of free HCl in 2 cases, seven and eight months of age, respectively.

(4) Pepsin was present in only 6 cases.

(5) Rennet was demonstrated in all but 2 cases.

(6) Lactic acid was present in all but 3 cases.

These data differ in many respects considerably from those recorded in previous studies. One cannot escape the impression of the noticeable lack of uniformity in the results yielded by most of the previous investigations, and of the lack of standards available for clinical purposes. It remains for future studies to place the clinical value of gastric analysis of the infant on as firm a basis as is that of the adult.

I take this opportunity of thanking Dr. Koplik for his encouragements, suggestions and material, also Dr. Asa B. Davis and Dr. R. W. Lobenstine for the use of their material at the New York Lying-in Hospital; Dr. S. Bookman for the examination of the gastric contents of the bottle-fed infants at the Physiological-Chemical Laboratory of the Mt. Sinai Hospital, New York City.

I am especially indebted to Dr. M. H. Kahn, interne at the Mt. Sinai Hospital, whose aid in connection with the examination of the cases has been of the greatest value during the whole course of study.

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THE RESULTS OF SUBSTITUTE FEEDING IN PREMATURE INFANTS.

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In this study of premature infants I am indebted to Dr. Rotch for permission to use the records of the Infants' Hospital and of the Children's Hospital. The managements of both institutions are planning to erect new buildings. One of the problems to be solved in connection with these plans is the provision which shall be made for the care of infants prematurely born. I have attempted to find out what have been the results of the present methods of treatment employed in the last fifteen years, hoping that some suggestions might be made for future requirements, or that some figures may be available for comparison with results obtained under new and better conditions.

The need for a well equipped ward for the reception and care of premature infants cannot be judged fairly by the number of cases which have been admitted in the past. With our limited number of beds many more cases are refused admittance than are accepted. There is no class of patients that requires more skilful nursing and careful feeding, and for the majority of poor, and even moderately well-to-do, people, the expense of proper home treatment is almost prohibitive. One is occasionally surprised to see how well a premature baby will do under simple home conditions with unskilled attendance; but the great majority of such cases among the poor in this country die from lack of proper care, whereas the statistics of certain foreign workers—notably those of Budin—show an extraordinarily low mortality among those who are reared in incubators and fed on breast-milk.

In this series I have purposely selected my cases in so far that I have excluded babies who entered the hospital so long after birth as to have passed the period of prematurity, as such infants belong naturally to the class of difficult feeding cases. I have also thrown out all cases in which there was definite evidence of diseases or conditions incompatible with normal development, such as congenital syphilis, spina bifida, congenital heart disease, erysipelas, etc. I have, however, included all those whose vitality had been greatly lowered during home treatment, who died soon after they were admitted to the hospital.

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The method of caring for premature infants has been practically the same at both the Infants' Hospital and at the Children's Hospital for the last fifteen years. The babies have been brought by the parents, without any supervision on the part of the hospital, and doubtless many of those who died soon after entrance had been improperly cared for at home previous to entrance, and were insufficiently protected from exposure on their journey from their homes to the hospital. With the exception of the period from 1895 to 1900 at the Infants' Hospital—to which I shall refer later—the babies have all been given oil baths, wrapped in quilted cotton jackets, and placed in padded cribs, with hot-water bottles to maintain the body temperature. The food has been almost without exception some form of modified milk; rarely a mother has supplied one or two feedings of breast-milk daily. The nursing has always been conscientious and skilful—the high mortality which has prevailed under this method of treatment cannot, I believe, be in any way attributed to lack of care. There has been no systematic isolation of the premature infants into separate wards, as our accommodations did not permit such an arrangement.

At the Infants' Hospital there were included in this series 95 cases, of whom 58, or 61 per cent., died; 24, or 25.3 per cent., lived and gained in weight; 13, or 13.7 per cent., lived but were discharged either losing in weight or making no gain. Of the last mentioned group many were taken home because of their condition and probably died, so that the ultimate mortality was undoubtedly considerably more than 61 per cent.

In the Children's Hospital there were 30 cases included in the series. Of these, 24 died, giving a mortality of 80 per cent., and 6 lived and gained in weight, a percentage of 20. The mortality was therefore nearly 20 per cent. greater at the Children's Hospital than at the Infants' Hospital. This difference is probably accounted for by the fact that no infant was discharged while losing in weight and condition. All were allowed to remain in the wards until their condition warranted treatment at home, or until they died.

The combined results of treatment at the Infants' Hospital and at the Children's Hospital, in reference to total mortality was as follows:—

Total Number of Cases.	Lived.	Per Cent.	Died.	Per Cent.
125	43	34.4	82	65.6

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When one compares this record with Budin's experience in four years' service at the Clinique Tarnier, in which 579 premature infants were treated with a mortality of only 10.2 per cent., one is impressed by the high mortality rate prevailing under a system in which breast milk has not been employed in the feeding. It is necessary to bear in mind, however, that Budin's very low mortality of 10.2 per cent. is due in part to the fact that the great majority of his premature infants weighed over 2,000 grams at birth; whereas, in the series here reported only 10 infants, or 9 per cent., weighed at birth over 2,000 grams. If we compare Budin's mortality in infants weighing from 1,200 to 1,500 grams, and again those weighing from 1,500 to 2,000 grams, with a similar series at the Infants' and Children's Hospitals the results are less unfavorable, although still inferior to Budin's experience. This is shown in the following table:—

TABLE I.

Weight.	Died.
1,200-1,500 grams	71.9 per cent., at the Infants' and Children's Hospitals.
" " "	60.0 " " at the Clinique Tarnier, 1898.
1,500-2,000 grams	44.2 per cent., at the Infants' and Children's Hospitals.
" " "	13.2 " " at the Clinique Tarnier, 1898.

As shown in the above table Budin's results were nearly 12 per cent. better than ours in infants weighing from 1,200 to 1,500 grams at birth, while in the class weighing from 1,500 to 2,000 grams his mortality was 31 per cent. better than was obtained at the Infants' and Children's Hospitals.

The mortality in all cases over 1,200 grams at birth treated at the Infants' and Children's Hospitals is shown in Table II.

TABLE II.

Showing the mortality in reference to the birth weight. When birth weights were not known the first weighing made after entrance to the hospital is assumed to be the birth weight.

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INFANTS' HOSPITAL.

Weight in Grams.	Living and Gaining Weight. No. Cases.	Per Cent.	Living but Losing Weight. No. Cases.	Per Cent.	Died. No. Cases.	Per Cent.
1,200 to 1,500	5	20.8	3	23.7	17	56.7
1,500 to 1,800	11	45.8	3	23.7	5	16.7
1,800 to 2,100	6	25.0	5	38.3	7	23.3
Over 2,100	2	8.4	2	15.3	1	3.3
		100.0		100.0		100.0

CHILDREN'S HOSPITAL.

1,200 to 1,500	1	16.7			6	37.5
1,500 to 1,800	4	66.6			6	37.5
1,800 to 2,100	1	16.7			2	12.5
Over 2,100	—	——			2	12.5
		100.0				100.0

In this series of cases no infant survived who weighed less than 1,200 grams. Infants weighing from 1,500 to 1,800 grams on entrance showed the greatest viability, judged by the percentage that lived and gained in weight. The explanation probably lies in the fact that the smaller the baby the sooner it was taken to the hospital after birth, the larger babies often being kept at home for two, and even three weeks, or until the failure of home treatment became manifest.

TABLE III.

Showing mortality in reference to the period of gestation.

INFANTS' HOSPITAL.

	No. Cases. Living.	Percentage.	Died.	No. Cases. Percentage.
Unknown	4	10.8	12	20.
6 months	0	——	8	13.
7 "	21	56.7	34	58.
8 "	12	32.5	4	00.7

CHILDREN'S HOSPITAL.

	No. Cases. Living.	Percentage.	Died.	No. Cases. Percentage.
Unknown	——	——	2	6.6
7 months	6	20.	21	70.
8 "	——	——	1	3.4

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Two deductions of value only can be drawn from the above table. First, in neither hospital did any infant in the sixth month of gestation survive; and, second, the percentage of infants in the seventh month who lived was nearly twice as great as those of the eighth month. It should be stated in this connection that the number of eighth month babies was comparatively small, and it is doubtful if the data as to the period of gestation can be relied upon, owing to the lack of dependence to be placed upon the statements of the parents in the class from which the infants were taken. The facts are consistent, however, with the view generally held, that premature babies of seven months are more likely to survive than those born prematurely at eight months.

In connection with the weight of a premature baby in relation to its viability, the following table is of interest, showing the length of time extremely small infants may live:—

TABLE IV.

1.	Weight—Grams.	Age at Entrance.	Lived after Entrance for
1	570	4 days	2 days
2	670	2 hours	6 hours
3	680	11 days	3 days
4	690	15 hours	48 hours
5	835	14 days	24 "
6	890	12 hours	27 days
7	930	24 "	8 "
8	940	17 days	7 "
9	940	9 "	2 "

In regard to the use of incubators, our experience is very limited. From 1895 to 1900 it was the custom to place most of the premature infants at the Infants' Hospital in an incubator, but the hospital records are not explicit upon this point, and one cannot be sure how many were treated in padded cribs by preference. The mortality during this period was in 25 cases 80 per cent.; whereas, from 1900-1905, when the incubator was discarded, it dropped in 43 cases to 58 per cent., and in 1905-1910 to 46 per cent. Some conclusions as to the advantages or disadvantages of incubators over padded cribs might be had by comparing the results obtained in hospitals in which padded cribs are used, and where breast milk is given as food, with our mortality in Boston, where padded cribs and modified milk have been

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the method of treatment. Unfortunately, I have no such data for comparison. It does not seem possible to draw any positive conclusions in regard to the relative merits of incubators and padded cribs from the temperature charts, as there is obviously a limit to which outside heat can be depended upon to maintain the internal temperature of the body, when the cause of variations in bodily temperature lies in disturbed metabolism. If general impressions are of any value, one would infer from the temperature charts of the cases studied that the method of padded cribs and heaters is efficient in providing the necessary outside heat; at the same time it has the distinct advantage of allowing one to regulate to a considerable extent the moisture and temperature of the air which is inspired, and greatly simplifies the problem of nursing. High as our mortality at the Infants' Hospital is today, it is distinctly lower than in the years when an incubator was in use. At the same time one must admit that we have had no experience whatever at either hospital with a modern incubator.

There does not seem to be any reason to believe that the inspired air should be at the same high temperature as that with which the body is surrounded. This is the objection which may be raised to all incubators. On the other hand, the padded crib, as we have used it, does not provide sufficient circulation of air, and one cannot help feeling that the baby breathes in too much of its own exhalations.

The incubator chambers—such as have been constructed by Bosi and Guidi in Venice, and Escherich in Graz—have much in their favor. The temperature and moisture of the air can be regulated in reference to the conditions suitable for aeration. But as different babies require different degrees of temperature to prevent the loss of body heat, further measures must be adopted to prevent loss of heat in individual cases. It would seem as if the thermo-pad, recommended by Dr. Holt, especially if made in the form of a sleeping-bag, would make it possible to keep several babies in the same incubator chamber at different temperatures, without altering the quality of the air which they breathe.

There is the utmost need of some convenient and expeditious way of transferring the patient from the house to the hospital without undue exposure. The wretched condition in which some babies arrive has convinced me that one cannot trust to the parents to provide suitable means of bringing the baby to the hospital without undue exposure and loss of vitality, especially in

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cold weather and when the distance is great. The best of hospital nursing and feeding is made useless in the end simply by the journey to the hospital. It should be the duty of the hospital itself to supervise the transportation of the baby. This can be arranged without an elaborate or expensive ambulance service. I would suggest that a portable receptacle should always be in readiness to send by express or by an automobile to the infant's home at the time the admission to the hospital is granted. The delay in sending the receptacle would be more than compensated for by the better condition of the baby when it arrived. A well padded basket with a cover, lined with hot water bottles, and containing a quilted gown in which to wrap the baby, could easily be provided and would serve its purpose. We have had no provision of this kind in Boston. The means of keeping the baby warm, and at the same time providing air to breathe, have been left to the parents. Judged by the condition in which many babies have arrived, the results have been deplorable.

Of the 82 cases who died, 20, or nearly one-fourth of the number, died within twenty-four hours, 7 within two days, and 11 within three days. Thirty-eight died within one week, 6 within two weeks, 6 within three weeks, 9 within four weeks, and 19 at varying periods over four weeks.

The possibility of materially lowering the mortality by providing proper means of conveying premature infants to the hospital would therefore seem to be very great.

The babies who died were on the average ten days old when admitted, and averaged 1,547 grams in weight. Those who lived and gained in weight were sixteen days old and averaged 1,750 grams in weight. Those who lived and lost in weight were seven days old and averaged 1,847 grams in weight. Judged by averages, therefore, those who were kept longest at home before being brought to the hospital were the ones who responded most readily to treatment. This may possibly be due to the fact that those babies were fed on breast-milk for longer periods and had a better start when it came to the point of providing substitute feeding.

The effect of substitute feeding in the care of premature infants must be judged not only by the mortality statistics, but by its influence upon the weight development. It is very clear, I think, from the results which I have tabulated, that the mortality under a system of substitute feeding is much greater than in

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cases in which breast-milk is used for food. An interesting question arises as to how favorable were the results of substitute feeding in the cases discharged as well and gaining in weight.

The average gain in weight of the 6 infants discharged as well from the Children's Hospital was 45 grams per week, practically 1½ ounces. At the Infants' Hospital, in 24 cases, the average weekly gain in weight was 50 grams, practically 1⅔ ounces. This represents very fairly the rate of growth one may expect in premature infants who survive and who are fed under the most favorable conditions of modified milk feeding for the first two or three months. The average number of days each child remained in the hospital was seventy. The cases were, therefore, under observation long enough to allow one to draw conclusions as to the effect of substitute feeding.

It is interesting to note the caloric value of the food given in relation to the gain in weight. For this purpose calculations of the energy quotient have been made in 11 of the cases that lived and gained in weight. In 6 cases, in whom the weekly gain was practically the same, the energy quotients varied from 72 to 137. One infant, taking a food which gave an average energy quotient of 72, made an average weekly gain of 73 grams. Another, with an energy quotient of 79, gained only 2 grams a week. The absence of any definite relation between the energy quotient and the gain in weight is very striking, as shown in Table V.:—

TABLE V.

Showing the average energy quotient in 11 infants who lived and gained in weight.

	Average Energy Quotient.	Average Weekly Gain in Grams.
1.	137	68
2.	133	77
3.	85	70
4.	92	70
5.	72	73
6.	127	68
7.	129	91
8.	79	2
9.	163	47
10.	80	34
11.	80	29

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The average energy quotient in these 11 cases who survived and gained in weight was 107.

The energy quotients in 18 cases that died at the Children's Hospital were also calculated, and are tabulated as follows:—

TABLE VI.

	Average Energy Quotient.	Average Weekly Gain or Loss in Weight in Grams
1.	26	— 40
2.	64	—119
3.	81	—159
4.	27	— 13
5.	79	—149
6.	39	—126
7.	66	+ 20
8.	28	— 25
9.	71	+ 20
10.	32	— 60
11.	70	—119
[illegible]	54	— 45
	80	— 53
14.	150	— 42
15.	65	—301
16.	109	— 43
17.	98	+157 (?)
18.	60	— 43

One might infer from the above figures that the infants in this group were, as a whole, underfed; yet there is no relation between the energy quotient and rate of loss in weight. Cases numbered 1, 12, 14, 16, 19, all lost from 40 to 45 grams per week, and their energy quotients were respectively 26, 54, 156, 109 and 60.

In some cases the very low energy quotient is to be explained when one understands that all infants on admittance were put on a very weak food and small quantities. Those that died within a few days or weeks could not be worked up to the average energy quotient of those infants who lived for two or three months.

Briefly stated, one may draw the following conclusions from

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the study of 125 premature infants, treated without incubators and fed upon careful modifications of cow's milk:—

(1) The total mortality was 65.6 per cent. in 118 out of the 125 babies weighing under 2,100 grams at birth.

(2) No infant weighing under 1,200 grams at birth survived.

(3) No infant in the sixth month of gestation survived.

(4) The smallest baby admitted weighed 570 grams at birth and lived 6 days.

(5) The possibility of materially lowering the mortality by providing proper means of conveying premature infants to the hospital is very great.

(6) Premature babies who survived gained on an average only 50 grams a week on modified milk, for an average period of 70 days each.

(7) The average energy quotient of the food of 11 cases who lived and gained in weight was 107.

(8) There was no definite relation between the energy quotient of the food and the weekly gain in weight.

(9) Modified milk, however, carefully administered and supervised, must be considered an unsatisfactory food for premature infants, and should be used only when breast milk cannot be obtained.

(10) Hospitals planning to receive premature infants should make ample provision for the maintenance of wet nurses.

(11) The relative value of padded cribs and incubators could not be accurately judged, as modern incubators have not been used at either the Infants' or the Children's Hospital in Boston.

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A COMPARISON IN BOYS AND GIRLS OF HEIGHT, WEIGHT AND EPIPHYSEAL DEVELOPMENT.

BY THOMAS MORGAN ROTCH, M.D.,
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The following remarks should not be understood to be a thorough digest of development in boys and girls, but merely the placing on record of certain observations which I have noted in my work with the Roentgen Ray on epiphyseal development.

As Professor Pryor, of Lexington, Ky., has come to the same conclusions from his work on the epiphyseal development of the two sexes, I think that at least it is worth while to present to this Society some of the practical deductions which may be made when we appreciate how, from anatomic differences in the sexes, boys and girls should be treated according to their anatomic development rather than according to their chronologic age.

It has long been known that while boys are usually heavier and taller at birth than are girls, and that the girl, according to the zone in which she lives, overtakes in height and weight the boy of the same age at the period of puberty, and surpasses him and becomes a developed woman before the boy becomes a man, when he again overtakes and surpasses the woman. It is, of course, well known that these periods of growth vary in the individual according to race and social conditions.

These rough charts, without attempting to illustrate the finer details of height and weight in feet and pounds, merely show how the lines of weight and height cross each other at about the same time, the twelfth to thirteenth year, but these ratios apply to boys and girls living in the same territories and of the same race and social condition. These lines of weight and height are shown so well by Camerer that I have taken his charts as illustrative and to compare with my own chart of epiphyseal development. The girl's line is shown by a broken and the boy's by an unbroken line. The girl's weight line is seen to cross the boy's at about the thirteenth year in this especial set of individuals, and the boy's to again cross the girl's at about the seventeenth year. In like manner the lines of height show about the same thing, but it must be remembered that they represent individuals of like surroundings and race. The girl's line is under the boy's until about the twelfth year and then crosses it and keeps above it up to about

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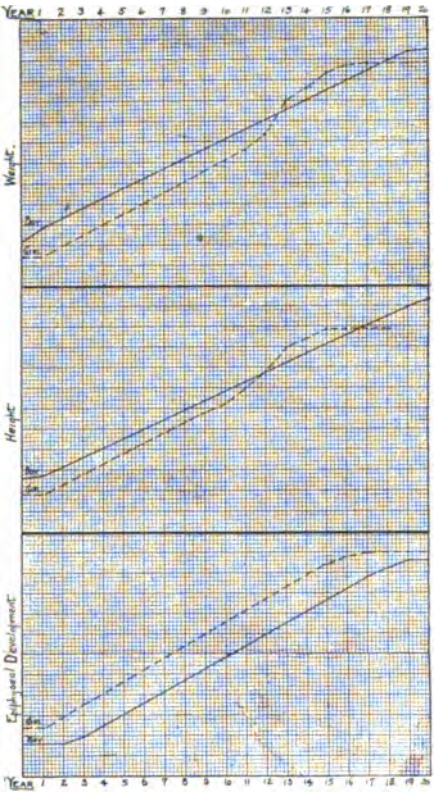
the sixteenth year, when the boy's line surpasses the girl's and remains above it.

A very large number of cases have shown that mere age does not necessarily determine weight and height, but only correlates when factors such as race, family, social condition, mode of life and climate are taken into consideration, and that these lines only



Charts showing relationship between boys and girls in regard to weight, height and epiphyseal development.

hold good under certain groupings and often do not in individual cases.

Still we can say, as a general rule, that girls are lighter and shorter at birth and that they remain so until about the age of puberty, when they surpass the boys and attain their mature weight and height some years earlier than do the boys. Boys, however, on becoming mature, again surpass the girls in height and weight.

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On examining my table, made up from over 500 cases, and also on referring to hundreds of others, I found that there was a decided difference between the epiphyseal growth and that of height and weight. I shall merely refer to the fact that my Roentgen work has shown very conclusively that the development of the carpal bones and the epiphyses of the hand and wrist correlates with that of the epiphyses elsewhere, also that this development can serve as an index for classifying individuals from birth to the completion of development. Following this study it is seen that individuals fall into groups, not chronologically, but by a graded set of developmental epiphyses.

Now if we examine this index still more carefully we will find that not only the groups which correspond in epiphyseal development differ quite widely in age, but that it is very apparent that the epiphyseal development of girls is in advance of that of boys from the very first year of life and remains ahead up to the time when the epiphysis, for instance that of the radius, becomes fully united. This unification, according to Pryor, takes place at about the sixteenth to the seventeenth year in girls and about two years later in boys. This means that the girl becomes a mature woman anatomically earlier than the boy becomes a mature man. My lines show this priority of the epiphyseal development in the girl over the boy from the first year of life, and are in striking contrast to the lines of weight and height.

These charts enunciate strongly the lack of correlation between epiphyseal growth and general physical development. When also we consider how often the epiphyseal growth seems to have a certain bearing, almost of correlation, on the mental growth, we can perhaps explain certain mental characteristics of girls as compared with boys. This again leads us on to the idea which I have expressed in previous papers, that in grading individuals from birth to the completion of development, the epiphyseal development should play a prominent part and should replace a chronologic grading; also that the child's general welfare depends to a certain degree on keeping its mental and epiphyseal development in equilibrium, thus avoiding overstrain, both mental and physical. This also leads up to the possibility that in grading the two sexes we should either have a separate curriculum for the sexes or a curriculum adapted to certain stages of epiphyseal development.

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THE FIRST HEART SOUND IN CHILDREN.

BY F. FORCHHEIMER, M.D.,
Cincinnati, O.

If one were inclined to believe that, after so long a time in which the normal heart sounds have been studied, there is a consensus of opinion among authors in regard to their production, a review of the literature would be disappointing. Indeed, in many things connected with heart diseases the statements of various authors are so much at variance as to seem almost incredible; one need only look at the descriptions of compensatory changes in valvular diseases to verify this.

In connection with the normal first sound of the heart there still exists much difference of opinion; but upon the whole certain modes of its production are accepted by the majority of authors, many of them differing as to details. As will be seen, I have preferred the term sound to that of tone, as the vibrations which we normally perceive over the heart are not sufficiently regular to produce a tone. And yet any one with a trained ear for music can determine the pitch of the first sound. This pitch depends, as I believe, not upon an overtone due to a combination of all the factors which go to make up the first sound of the heart, but upon its most important factor. In children the pitch is high, depending upon the age and size of the child, from *g* or *a* soprano below the staff, gradually becoming lower, until in a large male adult it reaches double *d* soprano below the staff (Landois).

The dominant factor in the production of the first sound is the systolic contraction of the myocardium. Next to this in importance is the vibration produced by the tense auriculo-ventricular valve; then those vibrations occurring in the large vessels when they are made more tense and lastly, and a little later, by the vibration of the semilunar valves. All this takes place during

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systole and all of it has been proven by clinical experience, so that instead of accepting only two factors for each side of the heart, four in all, we are forced to accept four for each side, *i.e.*, eight as the number of component parts of the first sound of the heart.

These then are the factors which enter into the production of the first sound of the heart, but specifically of the adult, as to duration, sound quality and accent or rhythm. In the child heart the conditions are still more complicated. As we are dealing with an organ slowly developing from the condition immediately after birth to that of adult life, there must, of necessity, be transitory and transitional phases which depend upon certain periods of development, broadly stated as existing from birth to the completion of the third or fourth year, from that time to sexual development and, finally, the adult period. But this manifestly holds good only as a general rule, to which many exceptions are found in individual cases. The first and probably the most important factor involved in the production of these changes is found in the size of the myocardium, which can be expressed by the ratio of the weight of the heart to the weight of the whole body. The following figures taken from Vierordt (*Daten. u. Tabellen für Mediziner,* 1906) show us that at birth it is 0.76 of the weight, gradually going down to 0.46 at the completion of the first year, then ascending until the end of the third or fourth year, when it reaches 0.53, then again falling to the tenth year to 0.41, gradually ascending to a maximum of 0.51 at the completion of the seventeenth year. The conclusion that may be drawn from this, as to the first sound, is that wherever the ratio between myocardium and body weight is great, the first sound will be strong, wherever it is low, it will be relatively weak—this I believe to have been able to verify by experience.

But there are other factors besides the size of the myocardium which must be considered in children in connection with our subject; not the least important being the question of elasticity of the myocardium, which has not received sufficient attention from clinicians so far as children are concerned. Melnikow-Raswedekow (*v. infra Fahr*) first showed that in the newly-born, elastic fibers are present only in the peri- and endocardium in the neighborhood of the large blood vessels, and none around the muscular elements of the myocardium, the latter being found only in adults. Fahr, who took up the subject again (*Virchow's Archiv.*, Bd. 185, XVIII., Vol. V., p. 29), verifies these statements, to

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which he adds the following: As a rule, the elastic tissue develops around the muscular cells about the fifth year of life and is completed at the end of the seventh year. In view of relations of weight of heart to weight of body, referred to above, it is interesting to note that increase in elastic tissue and increase in weight seem to occur at about the same time of life. The normal function of this elastic tissue is to cause dilatation of the heart, and in the adult diastole is due principally to this cause. In the newly born the elasticity of the muscular cells alone seems sufficient, and at the fourth year there begins the action of the elastic tissue and muscular elasticity which finally gives way to the preponderance of elastic tissue diastole by the end of the seventh year. Diastole increased by the elastic tissue affects the first sound, as the greater it becomes the more blood flows into the ventricle during a given time, the greater will the sytole become, and the more marked the first sound. To those who believe in the myogenic theory of the heart's action, all changes in rhythm are due to the automatic properties of the myocardium. However this may be, in children we normally find the infantile type of arrhythmia, in which the intensity and duration of the first sound are changed. Beyond this it is not my intention to enter into the discussion of the first sound in this connection, but not being able to accept the myogenic theory as explaining all, as Engelmann and others have put forth, it becomes necessary to call attention to some of the effects of the nervous system upon the first sound of the heart in children.

As Soltmann first showed, the great difference between nervous reaction in young and old animals lies in inhibition; in the young it is slightly developed, in the old more or less perfectly. This in children means a rapid pulse rate due to lack of cardiac inhibition; the difference in pulse rate in an infant awake and asleep is an example of this mechanism; but the difference in the first sound of the heart is not less remarkable; short, sharp, almost accentuated in the infant awake; longer, duller and not accentuated in the infant asleep. Gradually these differences disappear, but in certain individuals they last for a long time, especially in neurotic children.

Finally, the absolutely small weight of the heart must be mentioned—from 17.2 gm. in the first months to 41.2 gm. at the conclusion of the first year; 130.0 gm. at ten years as compared with 311.1 gm. at twenty-two years in the male and 253.5 gm. in the female (Vierordt), representing a sevenfold increase in

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weight in adult life. It must be admitted that if our premises as to the formation of the first heart sound are correct, a small vibrating mass will produce an altogether different sound from that of a large one.

All the various factors mentioned may, under proper circumstances, be followed by a change, the most important of which is called a bruit or a murmur. The division which Sahli makes of these has always appealed to me; it is as follows: (1) Organic and functional valvular murmurs. (2) Accidental murmurs. Both of these classes are of so much importance in children that a more or less detailed description is necessary. Whoever undertakes to make a diagnosis of a heart lesion in a child under three or four years of age in an offhanded manner and without due care and consideration, immediately stamps himself a dilettanté in heart diseases. Even the detection of bruits and their relation to the sounds may become difficult in these cases, due to the rapidity of succession of sounds, the peculiar rhythm in infants, and the intractability of children at the age of three or four years. But even when a bruit with the first sound of the heart *is* found, it is difficult to make out its significance. Traube first made the statement that the diagnosis of mitral insufficiency is the most difficult of all valvular diseases; the systolic bruit may follow an organic or a functional condition, or even be accidental. In addition the accentuation of the second pulmonary sound is of little value for diagnostic purposes, first, because it so frequently occurs in children who have no heart disease, and, secondly, it exists in both organic and functional valvular conditions in older children. In older children it frequently is of no value in accidental bruits. Moreover, in children it is difficult to say positively whether this systolic bruit is stronger over the usual auscultatory locations of the pulmonary or the mitral valves. It is true, we may be guided here by its conduction and frequently, but not always, by compensatory changes. Organic valvular systolic bruits acquired after birth are found in children at the apex in peri- and endocarditis, in mitral and tricuspid insufficiency, at the base in aortic stenosis, rarely in pulmonary stenosis. In congenital cardiac diseases they are found in pulmonary stenosis, stenosis of the mitral and tricuspid valves, defects in the ventricular septum and patency of the ductus arteriosus. An easy way for differentiating organic from other valvular bruits in children under three years of age has been given—one might almost say with a stroke of the pen—

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by Hochsinger. He says that under three years of age all bruits in the heart are organic. It is strange how widely this statement has been accepted. Even Romberg, in his excellent book (*Die Krankheiten d. Herzen u. d. Blutgefässe,* 1906, pp. 52, 185 and 220) comes back to it again and again and bases absolute differential diagnosis upon it. For the complete literature on the subject up to 1906, reference may be had to the excellent paper of Hamill and le Boutillier, which was read before this society—"Inorganic Late Systolic Pulmonary Murmurs in Infancy and Childhood" (*American Journal of Medical Science,* 1907, Vol. CXXXIII., p. 55-66). I fully concur with these authors in the view they have expressed. If we disregard the reasoning employed by Hochsinger, experience will have taught us that in children under three years functional and accidental bruits are found very commonly. Especially is this the case in infants, and principally systolic bruits. A further statement is made to uphold Hochsinger's view that organic bruits disappear because the lesion in an affected valve may disappear during childhood or later. This assertion cannot be denied, as we all have seen what we considered organic bruits disappear, but this is extremely rare, especially when the frequency with which bruits come and go in children's hearts is taken into consideration.

The systolic functional valvular murmur—and here we refer only to the auriculo-ventricular valves—usually depends upon a distention of the orifice of their insertion, followed by a relative insufficiency caused by an impossibility of complete restraint by the papillary muscles. They are very common in children and depend largely upon myocardial conditions. It is difficult to say which form of myocardial disturbance most frequently produces systolic bruits at the apex. If the statement of Schmaltz (*Zur Kenntniss der Herzstörungen in Scharlach u. ihre Folgen. Münch. Med. Woch.,* 1904, p. 32), that the most frequent heart lesion in scarlatina is an acute myocarditis and not endocarditis is true, in which he agrees with Romberg, we must consider acute myocarditis as the most common cause. This observer found, upon postmortem examination of scarlatinal patients, only 3 cases out of 33 which had endocarditis. Romberg (*Erkrankungen des Herzmuskels bei Typhus, Scharlach u. Diphtherie, Deutsches. Archiv. f. Klin. Med.,* Vol. XLVIII., p. 387) found, upon postmortem examination (method of Krehl) in 11 cases, 8 with myocarditis, 8 with pericarditis and 5 with endocarditis. We also find

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systolic apical bruits with the same mechanism in all other forms of acute myocarditis, due to diphtheria, typhoid, rheumatic fever, variola, gonorrhea and septicopyemia. For reasons that are self-evident they are also found in acute nephritis. I have seen a number of children who with every attack of high fever have a systolic bruit at the apex which grows louder with increase, and fainter with decrease, in temperature, which does not exist when the patient is well. One of my patients has been under my observation for over twenty years; he has grown to manhood and his heart seems to be perfectly normal. Another one, of the same class, had, when he was fifteen years of age, an acute dilatation of the heart, due to tennis, from which he, after eighteen months and now at the age of thirty years, also seems perfectly well. Schmaltz, in the article referred to, calls attention to a febrile bruit in scarlatina. But it would seem that the production of the bruit in the 2 cases just mentioned might depend upon different causes; in the first case it might be due to increase in expulsion of blood combined with low blood pressure (an accidental bruit); in the second it is possible that there was a slightly damaged heart from acute myocarditis, especially as the boy had had rheumatic fever.

In chorea the bruit is produced by acute endocarditis. Possibly it may be an accidental bruit, and by some it is supposed to be a disturbance in innervation of the papillary muscles or of the myocardium itself.

In acute dilation of the heart due to excessive use, especially when the heart is in a transitory period of growth, the bruit is frequently found. I have in a few instances of chorea of the grave form also found enlargement of the heart, with systolic, as well as with diastolic, bruits.

The accidental bruits which are not produced in the valves and which exist without apparent lesions are many in children. We do not err when we say that the mechanism of their production is not satisfactorily explained, certainly there is no general consensus of opinion on the subject. The one most commonly found in children is the systolic bruit over the pulmonary valve, to which attention has been again called by the articles of Hamill and le Boutillier (l. c.) and Lüthje (*Zur Physikalischen Diagnostik am Herzen, Speziell üb. Systolische Geräusche an d. Valvulamitralis u. Pulmonalis, Münch. Med. Woch.*, 1907, Vol. LIV., p. 49), both of these articles appearing at about the same time. The first-

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named authors report 80 cases and the latter one has found it present, alone or combined with a mitral systolic bruit, in 612 children out of 854 which he examined in Erlangen. It is systolic, usually late in systole. Hamill and le Boutillier state that it is superadded to the first sound, occupying the greater part of the systolic interval, but more frequently its early portion. It is best heard over the pulmonary, but may also be heard over the mitral valve. It is best heard in the recumbent position, at the end of inspiration, disappears upon forced inspiration and increases upon forced expiration. It may be transmitted and conducted to the vessels in the neck. There is no compensatory hypertrophy.

Many explanations have been offered for this bruit, which have been given in the first of the two articles just mentioned. Lüthje (l. c.) has added another one; he claims it to be not impossible (*nicht unwahrscheinlich*), that it is a pulmonary stenotic bruit. From his report alone one is not able to decide whether an ordinary stenosis or some other form is referred to. The only conclusion one can arrive at in this regard may be obtained from the remarks which Schott makes in the discussion, when he says he does not believe this bruit due to change of posture. As Lüthje does not describe any compensatory changes, it is not reasonable to suppose that so excellent an observer would consider the bruit, which never has the character of an organic stenotic bruit, as due to changes in the valve. Moreover, it would be unreasonable to imagine that so many cases of organic pulmonary stenosis exist as reported here by so many authors, which disappear entirely. It has seemed to me that if the development of the elastic tissue in the myocardium, referred to before, is taken into consideration, the bruit might also be explained. During diastole the elastic fibers, which are present in the youngest children around the large vessels, on account of their elasticity contract away from the lumen of the conus arteriosus and the vessels; during the systole they are crowded back by the contraction of the myocardium. If there be a disparity between the strength of the myocardium and the resistance of the elastic fibers, there will result more or less of an inequality in the conus arteriosus, which acts as an obstacle. When the blood passes over this a bruit is produced. This bruit must of necessity be heard most frequently and most intensely at the end of systole, for it is then that the greatest force of the myocardium is expended at a given place, just as in mitral stenosis this increased force at the same time causes greater

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velocity of blood current. But the relative disparity may be so great as to cause an obstruction during the whole of the systole, then the bruit develops early in systole which, in fact, is the more frequently the case the younger the child. On the left side of the heart this condition cannot exist, as the left myocardium is already so much more developed, so that the elastic hindrance is easily removed. In younger children it is, therefore, more frequently heard, but in accordance with this explanation the bruit may be produced and is produced until the adult heart develops, as late as eighteen years.

As far as the other systolic accidental murmurs are concerned, as Sahli says, "their real origin still remains unproved." An exception must be made for the cardiopulmonary bruit, rare in children, whose origin is self-evident. For the origin of the others many things have been considered—the irregularity of the inner surface of the heart, the possibility of temporary obstruction at the place of junction of the arteries with the ventricles, the rapidity of the blood current, the quality of the blood (possibly its viscosity), the diastolic filling of the ventricles and the rapidity of expulsion, the condition of the myocardium, the peripheral blood pressure and nervous conditions. Under these conditions, as Sahli suggests, it is easier to say why a bruit should be present than absent.

The anemic bruit can, then, be easily explained; that it is directly produced by anemia is a matter of some doubt. Certain it is that in many cases of anemia there is no bruit, and many bruits are attributed to anemia which are due to other causes. On the other hand, cases of oligochromemia of a severe type, below 25 per cent. Hb., usually have accidental bruits, it matters not what the form of anemia may be. It is certainly impossible to diagnosticate anemia by listening to the heart, and while the "anemic bruit" must still be retained in the present state of our knowledge, the general condition must be first known, as well as all other causes, for the elimination of this bruit. The same may be said for the bruits in general debility and in fever. All other bruits must be excluded before the accidental bruit is accepted in these conditions. Indeed, the careful diagnostician does this in every case, but with all precautions taken an error in diagnosis may, and sometimes does, occur.

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DISCUSSION.

DR. NORTHRUP.—I have enjoyed the explanation of this common phenomenon very thoroughly and I would emphasize the fact that many murmurs from unexplained causes disappear. Hochsinger's first statement has held the stage until now; it holds it now I think. Ten years ago I published the statement that at the New York Foundling Hospital I had not found cases of organic lesion in children under three years with one exception. Frequently others have taken the same ground. I should like to hear it emphasized now that such murmurs do exist and disappear without any remaining lesion.

DR. HAMILL.—I have been much interested in the explanation which Dr. Forchheimer has given, accounting for the production of these murmurs. In the paper of Dr. Le Boutillier and myself, which has been referred to, we offered no new explanation for the occurrence of these murmurs, but we accepted as the most reasonable theory that had been advanced one which had been considered for a good many years and which had received recently some experimental confirmation at the hands of Drs. Thayer and MacCallum—that it was due to dilatation of the conus arteriosus and pulmonary artery, resulting from variations in pressure. It seems to me that Dr. Forchheimer's explanation is reasonable as well as valuable, and I am glad to have heard it, because the more I study these murmurs in early life, the more I am convinced that there must be more than one etiologic factor at work in their production. The murmurs vary enormously in point of maximum intensity; in distribution, in volume and in pitch. In the last few years I have heard more murmurs in the aortic area than in former years.

I am glad to hear Dr. Forchheimer express the view that Hochsinger's attitude toward these murmurs is incorrect. I have recently had occasion to make physical examinations of 100 children to eliminate any organic lesions of the heart, and in 38 successive cases we found some type of functional murmur present.

As to the effect of posture, I do not recall that Dr. Forchheimer said what his experience had been, but I am convinced that functional murmurs are often capable of demonstration when children are placed in the recumbent posture, which are not present when they are in the upright. Whether one should consider these postural murmurs I am not prepared to say. I think the change in position probably exaggerates the conditions which produce the murmurs which are heard when the child is upright.

I was very much interested in Dr. Forchheimer's statements regarding the first sound of the heart, as to the gradual change in quality of that sound during the first years of life. I have noticed the types of arrhythmia in nervous children to which he

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refers. and I fully accord with the opinion that the accentuation of the second pulmonic sound is of no value in differentiation.

DR. CHURCHILL.—I recently had a case which illustrates this idea—a child with nephritis, and absolutely nothing about the heart. Two or three days later it developed an acute arthritis of the knee joint. I watched the case every day. It developed a systolic murmur heard at the second interspace close to the sternum. Soon after it developed well marked precordial friction sound. Then the murmur began to gradually subside. Finally it came down to normal. Then the knee started up again and the temperature went up, but the murmur disappeared entirely. In a case like that it must be due to infection of a heart valve. Two blood cultures were absolutely negative.

DR. FORCHHEIMER (closing).—We do not want to go to the other extreme and say there are no organic bruits in the heart in children under three years of age. I have seen these children loose the bruit as they grow up. I recall a case of a patient four years of age with an attack of rheumatic fever that developed a heart bruit, with chorea. The patient is now about thirty and that bruit has entirely disappeared; there remains no evidence of any myocardial or valvular trouble. We do see endocarditis in children in a large percentage of cases.

What I wanted to especially establish for children under three years is just the opposite of what Hochsinger states. I want to bring the matter to a concrete expression. The reason his statement has been accepted is because most of the work done on the heart has been done in connection with adults. A number of others, who perhaps have not had the material among children, have simply reprinted this statement. Those of us who see a great many children and hear these bruits know that they are frequently to be heard in children under three years.

As to the matter of posture, we have no explanation for that. It is the case with all systolic bruits that they increase in the recumbent posture.

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A STUDY OF THE VALUE OF BRUDZINSKI'S "NECK SIGN" AND OF THE CONTRALATERAL REFLEX IN THE DIAGNOSIS OF MENINGITIS IN INFANCY AND CHILDHOOD.

BY JOHN LOVETT MORSE, A.M., M.D.,

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Brudzinski, in a paper on "The Contralateral Reflexes in the Legs in Childhood," published in 1908 (*Wien. klin. Woch.*, 1908, p. 255), drew attention to a sign which he found in tuberculous and in epidemic meningitis, and occasionally in other conditions, but never in healthy children. This sign was a concomitant reflex of the leg on one side when passive flexion of the leg on the other side was made—the identical contralateral reflex. Sometimes the motion of the other leg was extension instead of flexion—the reciprocal contralateral reflex. He found the identical contralateral reflex in 6, and the reciprocal contralateral reflex in 1 of 8 cases of tuberculous meningitis, and the identical contralateral reflex in both of 2 cases of cerebrospinal meningitis. In some cases the reflex was present on both sides; in others, on but one. He also found it in a case of Little's disease. He concluded that in connection with other signs, such as Kernig's and Babinski's, it was useful in the differential diagnosis of meningitis. In a later paper (*Archives de Médecine des Enfants*, 1909, Vol. XII., p. 745) he states that further study has confirmed him in his conviction of the value of this sign, and that it has materially aided in the diagnosis of certain cases in which Kernig's and Babinski's signs were absent. He had then found it in 17 cases of tuberculous and 6 of cerebrospinal meningitis, the sign sometimes being present on both sides and sometimes on but one.

His explanation of this sign is, in brief, that there is a congenital tendency to bilateral innervation and that there is an anatomical connection between the centers of corresponding muscles. This connection is more definite and persists longer in the legs than in the arms because of the slighter differentiation of function in the former. When the cerebrum is diseased there is, in infancy and childhood, a tendency for the more specialized functions to revert to the younger type. The result of such reversion is the production of the contralateral reflex.

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Greco (*Revue d'Hygiene et de Médecine Infantiles,* 1909, Vol. VIII., p. 130) never found this phenomenon in healthy children or in those ill with diseases of the gastrointestinal tract or lungs. He did not find it in children with rickets or backward in development. It was also absent in 1 case each of acute infantile paralysis, spastic paralysis, chronic internal hydrocephalus, idiocy and tetany, and in 2 of chorea. Its absence was of considerable value in ruling out meningitis in several cases, which were at first supposed to be meningitis, but which later turned out not to be. He reports 10 cases of tuberculous meningitis in which this sign was present, and concludes that its clinical importance in the diagnosis of tuberculous meningitis in young children is indisputable. The diagnosis in some of these cases seems, however, from the data given, somewhat doubtful.

Zaimovsky (*Archives de Médecine des Enfants,* 1909, Vol. XII., p. 796) confirms these observations and says that the contralateral reflex is almost constant in children having an affection of the meninges.

Brudzinski, in 1909, published another paper entitled "A New Sign in the Legs in Meningitis in Childhood; The Neck Sign" (*Archives de Médecine des Enfants,* 1909, Vol. XII., p. 745), in which he stated that in meningitis passive flexion of the neck forward causes flexion of the legs at the hips and knees and a marked flexion of the legs on the pelvis. The test is performed by taking the head of the child, lying flat on its back, in the left hand and flexing it on the chest, the chest being held down by the right hand. He found this sign in 20 of 21 cases of tuberculous, in all of 11 cases of cerebrospinal, and in 2 of pneumococcus meningitis. The case of tuberculous meningitis in which this sign was not obtained was not examined until just before death. In one of the cases of pneumococcus meningitis the neck-sign was the only one of the usual signs of meningitis present. He also found it in 8 cases of serous meningitis, which developed in the course of different infectious diseases. Comparative studies of a series of children of all ages, some healthy and some ill with various diseases, failed to show this sign. It was present, however, in a case of "meningismus," in one of encephalomyelitis, in one of 5 cases of Mongolism, and in 4 infants backward in development.

A study of the relative frequency of the various signs of meningitis in 42 cases showed that the neck-sign was positive in

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97 per cent., the contralateral reflex in 66 per cent., Kernig's sign in 57 per cent., and the Babinski phenomenon in 50 per cent.

He is unable to give a satisfactory explanation for the origin of this sign. It cannot be due to increased cerebral pressure alone, because it is not found in hydrocephalus. Neither can it be due to spasm, because it is absent in Little's disease. It cannot be due to pain, because it is often present in meningitis when there is no pain, and absent in normal children in whom flexion of the head seems to cause pain. He concludes that in all probability the most important factors in its production are a muscular hypertonus of the legs and a physiologic predominance of the extensor muscles of the neck and back over the flexor muscles of the legs.

Since the neck-sign was a new one and this application of the contralateral reflex also new, and since so much importance was attached to them by the various writers in the diagnosis of meningitis, it seemed to me worth while to go over the ground again in order to form my own conclusions as to their value in this connection. I have, accordingly, examined 400 children, either well or ill with diseases other than meningitis, as to the presence of this sign. Ninety were well; 115 were suffering from diseases of the respiratory tract, including 14 cases of otitis media and 40 of lobar-pneumonia; 98 were ill with various diseases of the gastroenteric tract, either acute or chronic; 31 had various diseases of the nervous system other than meningitis and will be considered later. The remainder had a great variety of diseases—typhoid, nephritis, influenza, heart disease, rickets, purpura, scarlet fever, measles, and so on. Neither the neck-sign nor the contralateral reflex was elicited in any instance. The ages of the children varied from six days to twelve years. Ninety were less than a year old, 67 between one and two years, 102 between two and five years, and 141 over five years.

The diagnoses in the 31 patients ill with diseases of the nervous system were as follows:—

Cerebral tumor	1
Old spastic hemiplegia	1
Chronic internal hydrocephalus	3
Idiocy of various types	6
Acute anterior poliomyelitis	1
Acute cortical encephalitis	1
Diphtheritic paralysis	1
Facial paralysis	1

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Chorea	2
Habit spasms	2
Epilepsy	1
Eclampsia	11
	31

Five of the cases of eclampsia were examined during the convulsive state, the others in the intervals between convulsions.

It seems safe to conclude, therefore, from the study of these 400 cases, that Brudzinski and his followers are correct in their statements that neither the neck-sign nor the contralateral reflex are present in well children or in those ill with diseases other than of the nervous system, and that they are very seldom met in diseases of the nervous system outside of meningitis.

I have examined 11 cases of tuberculous, 5 of cerebrospinal and one of pneumococcus meningitis with relation to the presence of the contralateral reflex and the neck-sign. No attempt was made to compare the frequency of these signs with that of Kernig's sign, rigidity of the neck or the condition of the knee-jerk. The ages of the patients varied from six months to eleven years. The diagnosis in the tuberculous cases was proved by autopsy in one, by the finding of tubercle bacilli in the fluid from lumbar puncture in another, and by the presence of a fibrin clot, an excess of cells and a predominance of lymphocytes in the cerebrospinal fluid in the remainder; that in the cerebrospinal and pneumococcus cases by finding the causative organism in the cerebrospinal fluid.

Tuberculous Meningitis.—Owing to the conditions under which most of the patients were seen, it was impossible to make daily examinations. Some were seen but once; in others the examinations extended over a period of several days, in one instance as long as sixteen days. They were first seen, on the average, about ten days after the onset, while, as a rule, the last examination was made on the day of death, or one or two days earlier. There was nothing constant about the results obtained. The contralateral reflex was usually absent; when present, inconstant; sometimes present on one side only, sometimes on both sides; sometimes associated with the neck-sign, sometimes not. When present it was always identical. The neck-sign was found more often, but was never constant. In one case, examined daily from the tenth day of the disease until death, on the seventeenth day, it was never found; while it was always absent in another patient

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studied for three days near the close of the disease. When it was present, flexion usually occurred in both legs, but sometimes in only one. The number of observations is too few to warrant any definite conclusions, but both the neck-sign and the contralateral reflex were apparently more often present about the middle of the disease than at the beginning or just before death.

Cerebrospinal Meningitis.—The contralateral reflex was never found. The neck-sign was positive much more often than in the tuberculous cases. When it was present, flexion always occurred in both legs. In three instances it was constant throughout the height of the disease. In the three patients who recovered it disappeared during convalescence, and in one of them, seen a month later, it was still absent. Another patient was taken out of the hospital while it was still present. It was absent in the fatal case on daily examinations from the fifteenth day until death, on the twenty-first day. The rigidity of the neck not infrequently prevented a satisfactory examination.

Pneumococcus Meningitis.—This patient was seen but once, the day before it died. There was paralysis of the whole right side. The contralateral reflex and the neck-sign were both present on the non-paralyzed side.

Conclusions.—The contralateral reflex and the neck-sign are never present in healthy infants and children or in those ill with diseases other than of the nervous system. They are almost never present in diseases of the nervous system outside of meningitis. The contralateral reflex is found much less constantly than the neck-sign. Both may occur intermittently or be absent throughout the whole course of the disease. Their presence in an acute disease is strong evidence in favor of meningitis; their absence does not exclude meningitis. The diagnostic value of the contralateral reflex is less than that of the neck-sign. They occur in all types of meningitis and are of no importance in differentiating between them.

DISCUSSION.

DR. NICOLL.—"Three weeks ago, I saw a case of tuberculous meningitis in which the "neck sign" was positive. The child, when left undisturbed for an hour or so, would give a distinct reaction, but if tried at frequent intervals there was very much less of a response. I had an opportunity of testing this symptom also in a case of cerebrospinal meningitis and was able to get a very positive response, although it varied in intensity at different times.

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THE CYTODIAGNOSIS OF TUBERCULOUS MENINGITIS AND THE POSSIBILITY OF RECOVERY.

BY CHARLES HUNTER DUNN, M.D.,
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The patient, whose illness forms the basis of this paper, was a little girl three years of age, who was seen in consultation with Dr. Herbert W. Newhall, of Lynn, on December 7th, 1908. Her father, mother and a younger sister were well. A brother of her father had tuberculosis of the spine. The previous occupant of the house in which the patient lived died of tuberculosis of the lungs.

The patient was breast-fed until nine months old, and was well until seven months old, when she became subject to intestinal indigestion, which was equally severe while she was still on the breast and after weaning, and which continued for about seven months. Adenoids were removed in April, 1907. She had, since sixteen months old, been subject to attacks of diarrhea, with mucus in the movements. Between these attacks digestion had been normal, and she had seemed perfectly well. She had a mild attack of vomiting during the summer of 1908, which was attributed to indigestion. Two weeks ago she caught cold, and still had some cough, although the cold had been improving.

On December 5th, at 2 A.M., the patient began to vomit. She was immediately given small doses of calomel, and all food was withheld, but, in spite of this, vomiting continued all day, even the smallest amount of water being vomited. At 5 P.M. vomiting ceased. During the night she was fussy and irritable, but the next morning seemed better, and played a little. In the afternoon she vomited once, and did not seem so well. Since then she had grown increasingly more apathetic. Her temperature had been between 99° and 100° F. Her increasing apathy was the most notable symptom.

On physical examination her temperature was found to be 99°F., her pulse 74, and her respiration 30. She was a well developed, fairly well-nourished child. She lay on her back with her eyes open, and took very little interest in her surroundings, not responding when spoken to, although evidently understanding.

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She did not resist examination as she usually does. At times the expression of her eyes became peculiarly fixed and staring. There was slight ptosis of the left eyelid, and the pupils were equal and reacted normally to light. There was no trace of retraction of the head, nor of tenderness or rigidity of the neck, nor of tenderness about the ears. Breathing was slightly suggestive of obstruction in the nasopharynx, but was otherwise normal in character. The mouth, tongue and throat were normal. The heart was normal, except for a faint systolic murmur at the base. The pulse was slow, regular and of good quality. The lungs were normal, except for a few coarse, moist râles at the left apex. The abdomen, liver, and spleen were normal. The limbs were normal, showing no evidence of paralysis or spasm. The knee-jerks were both more lively than normal. The plantar reflexes were not obtained. There was a moderate Kernig's sign present on both sides. There was no ankle clonus.

It was decided that the case was strongly suspicious of tuberculous meningitis, and that the other possibility was cerebral symptoms from absorption of intestinal toxins. It was advised to give castor oil, a high irrigation of the colon, and water diet until the next day.

Toward the end of the visit the patient began to cry out at intervals as if in pain. Following these attacks of crying there was noted marked rigidity of the right hand and arm, with twitching of the right hand, which lasted several minutes.

The patient was seen again on the following day. She had had rather a bad night, with crying, and considerable twitching and rigidity of all the limbs, which at times amounted to clonic and tonic spasms. Bromides had been given to control these symptoms. She had had one normal looking movement. The enema obtained very little normal fecal matter. The temperature had not been above 100°F. She was refusing everything given by mouth.

On physical examination the apathy was more marked. The eyes were fixed, with a distinct cerebral look. There was occasionally slight internal strabismus, but the ptosis of the left eyelid was not visible. There was considerable spasm of the right arm and leg. The knee-jerks on both sides were markedly exaggerated, and Babinski's sign was present on both sides, most marked on the right. There was no ankle clonus, and no rigidity of the neck.

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Lumbar puncture was then performed and 10 c.c. of very slightly cloudy fluid obtained under considerable pressure. Examination of the cerebrospinal fluid showed no fibrin formation. The cell count showed 260 leukocytes per cubic millimeter. Differential count of the cells showed the following: Polynuclear cells, 2 per cent.; lymphocytes, 96 per cent.; endothelial cells, 2 per cent. In the centrifugalized sediment, with the Löffler's methylene blue stain, no microörganisms were found, but with the stain for tubercle bacilli several small clumps of acid-fast bacilli were found, which in appearance exactly resembled typical tubercle bacilli.

In view of this examination, a positive diagnosis of tuberculous meningitis was made, and an unfavorable prognosis given.

On the following day, December 9th, the patient was seen again. During the night she was no better, having been very apathetic, with marked rigidity and twitching of the limbs. The pulse was more rapid, and at one time during the night the respiration was rapid and irregular. The temperature went up to 101.4°F. In the morning, however, the patient became much better. The temperature came down to 99°F., the respiration became normal, and all rigidity, spasm and twitching of the limbs disappeared. The child was found in an apparently normal sleep, and was not disturbed for examination.

On December 10th the patient showed a very marked improvement. She was still somewhat apathetic, and showed a tendency to sleep a good deal. But she took at times an active interest in her surroundings, and responded well. She had slept all night, and showed no spasm nor rigidity of the limbs. Her temperature was normal, her pulse 104, respirations 30. There was no spasm or rigidity of the limbs, no ptosis nor strabismus, and no Babinski. Kernig's sign was still present, although not marked, and the knee-jerks were still very lively, though not so markedly exaggerated as on December 8th.

On December 11th the improvement had continued. The temperature was normal, and the patient was much brighter. There was no notable change on physical examination. From this day, until December 17th, there was no notable change, except that on the 16th she seemed less well, her pulse for a time becoming much slower, 78 per minute and slightly intermittent. It was noted that she did not use her right arm quite as well as her left. On the 17th she seemed very much brighter, and Kernig's sign disap-

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peared. Physical examination was normal, except for somewhat exaggerated knee-jerks. On December 21st the child seemed absolutely normal, except she had not yet talked. On December 28th she was considered perfectly well, and has been well ever since, except for a few mild digestive disturbances.

It is unfortunate in this case that the proof of the existence of tuberculous meningitis was not completed by injecting the cerebrospinal fluid into a guinea-pig. The possibility of recovery was not considered until a time when further lumbar puncture was not advisable. Nevertheless, the writer believes this case was actually a case of tuberculous meningitis, the evidence of the bacilli being almost absolutely conclusive, although the entire chain of proof was not complete.

The Fatal Prognosis of Tuberculous Meningitis.—Tuberculous meningitis is usually recognized as an invariably fatal disease, and an absolutely unfavorable prognosis is usually given. This fact causes this disease to be one of the most depressing both to the friends of the patients and to the physician. An absolutely unfavorable prognosis should not be given if there is the faintest hope of recovery which can be given to the patient's friends. The writer believes that even in tuberculous meningitis we are not justified in giving such a prognosis, but that we are justified in holding out some slight hope. My purpose is to bring forth what faint hope may be given in this disease.

The Diagnosis of Tuberculous Meningitis.—The diagnosis of tuberculous meningitis rests, usually, upon a triple basis; first, the clinical characteristics of the disease; second, the results of the examination of the cerebrospinal fluid obtained by lumbar puncture; and, third, the fact that death invariably occurs.

The clinical picture in this disease is admittedly inconclusive in establishing the correctness of the diagnosis. The writer has analyzed a series of 60 successive cases of tuberculous meningitis, the results of which analysis may be seen in Table I.

It appears that no one of the many characteristic symptoms and signs of the disease is either invariably present or absent, although various signs and symptoms which occur most frequently may be grouped together into our common conception of the typical case. From this typical case every sort of wide variation is frequently to be met with. The onset may be sudden or gradual, the temperature may be high, moderate or low; unconsciousness may develop rapidly and early, or slowly and late, and

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such symptoms and signs as headache, rigidity of the neck, Kernig's sign, knee-jerks, localizing neurological signs, convulsions, vomiting, leukocytosis, may all be either present or absent.

TABLE I.

Symptoms.	Number of Cases.
Onset	Sudden 9; Medium 13; Gradual 37
Temperature	High 11; Moderate 18; Low 19
Unconsciousness	Early 2; Medium 15; Late 41
Headache	Present 17; Absent 42
Rigid Neck	Early 7; Developed 29; Absent 23
Kernig	Early 7; Developed 21; Absent 31
Knee-Jerks	Exaggerated 15; Normal 20; Absent 24
Localizing Neurologic signs.	Early 2; Developed 19; Absent 39
Babinski	Present 19; Absent 35
Convulsions	Early 8; Developed 12; Absent 40
Vomiting	Present 41; Absent 19
Pulse	Slow 15; Rapid 43
Cheyne-Stokes Breathing.	Present 18; Absent 42
Leukocytes	Present 10; Absent 8
Tuberculin Reaction.	Present 8; Absent 5
Spinal Fluid	Clear 51; Slightly cloudy. 7; Cloudy 0; Bloody 2
Intradural Pressure.	Present 42; Absent 16
Predominating Cells.	Mononuclear 60; Polynuclear 0
Fibrin	Present 42; Absent 16
Bacilli	Found 15; Not found 45

TOTAL DURATION.

2— 4 days	3
5— 7 days	4
8—14 days	15
15—21 days	15
22—28 days	11
Over 28 days	8

The characteristics of the cerebrospinal fluid are of greater value than the clinical picture in the diagnosis, but are often inconclusive as the basis of a positive diagnosis. Were it possible to demonstrate tubercle bacilli in all cases, or even in the great majority of cases, the examination of the cerebrospinal fluid would be of positive value. But the difficulty of demonstrating tubercle bacilli in the fluid obtained by lumbar puncture is so great that it has become the usual practice to depend upon other characteristics of the fluid. The clearness of the fluid is a useful means of distinguishing the tuberculous from certain other forms of menin-

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gitis, although there may be slight cloudiness, but is of no value in differentiating tuberculous meningitis from other diseases. The main reliance is placed on the predominance in the fluid of the mononuclear lymphocytes, as shown by a different count of the cells in stained coverglass preparations. In the writer's analysis of 60 cases this predominance of lymphocytes was present in every case, and its absence should be regarded as precluding a diagnosis of tuberculous meningitis. Nevertheless, the writer has seen a number of cases in which such predominance of lymphocytes was present, and which were consequently diagnosed as tuberculous meningitis, but which later proved to be other diseased conditions. It has also been shown that the mononuclear lymphocyte is normally present in the cerebrospinal fluid. Reliance must consequently be placed upon a diagnostic increase in the number of lymphocytes in tuberculous meningitis. With the method usually employed in the examination of the cerebrospinal fluid, that of making coverglass preparations from the centrifugalized sediment, the lymphocytes present in normal fluid might easily, through centrifugalization, be made to appear increased in number. This probably accounts for many cases of mistaken diagnosis. Such a method of examining a centrifugalized sediment is far too inaccurate to demonstrate an increase in the number of lymphocytes. More accurate methods have been devised for demonstrating an increase in the number of lymphocytes, and a standard number of lymphocytes for normal fluid has been determined. But an increase in this number has been found in certain diseases other than tuberculous meningitis.

A third characteristic of the disease, the invariable occurrence of death, is frequently brought forward as a basis of confirming or rejecting the diagnosis of tuberculous meningitis. The outcome is most frequently used in rejecting the diagnosis in cases which, having been diagnosed as tuberculous meningitis, end in recovery. It is then argued that the diagnosis was wrong, because tuberculous meningitis is invariably fatal. No more perfect example of begging the question can possibly be imagined than such an argument, for it makes an invariably fatal outcome essential to the diagnosis of a disease, the invariable fatality of which can only be proved by the diagnosis. There is no especial reason other than experience for regarding tuberculous meningitis as always fatal. The analogy of other acute tuberculous processes

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is in favor of the possibility of recovery. There are on record many cases of recovery in which the diagnosis was supported by the clinical picture and the cytological characteristics of the cerebrospinal fluid, which, while not proven to be actual cases of tuberculous meningitis, were not proven not to be cases of this disease.

Evidence of the Possibility of Recovery.—It is seen, therefore, that the conditions surrounding the diagnosis of tuberculous meningitis are of such a character as to render the making of a positive diagnosis in this disease extremely difficult, and only to be attained in a comparatively small number of cases. It is impossible to prove that the disease is invariably fatal. It is quite conceivable that, with a very high, but not absolute, fatality, there might be no cases of recovery on record in which the proof of the correctness of the diagnosis was complete. Every case of apparent recovery in which absolute proof was lacking has been rightly rejected. The possibility of recovery is as difficult to prove as invariable fatality.

Nevertheless, some evidence of this possibility has appeared in medical literature from time to time. A few, very few, cases of recovery have been reported in which tubercle bacilli were found in the cerebrospinal fluid. If these few cases bear the same proportion to the whole number of recoveries as the number of cases in which diagnostic proof is completed bears to the whole number of cases, the possibility of recovery is sufficiently great to interest the clinician.

The Count of the Number of Lymphocytes per Cubic Millimeter in the Diagnosis of Tuberculous Meningitis.—Anything increasing the certainty of the diagnosis of tuberculous meningitis, or which increases the frequency with which a positive diagnosis can be made, will be of great future value in determining the question of the prognosis of this disease. In this connection the writer has turned his attention to the estimation of the exact amount of the increase in the number of lymphocytes seen in this disease, as compared with the number of lymphocytes seen in normal cerebrospinal fluid, and in other diseases, with a view to determining whether, in tuberculous meningitis, there is constantly a sufficient increase to constitute a diagnostic characteristic of this disease.

It is to Fuchs and Rosenthal that we owe the introduction of

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the more accurate method of slide counting, which has replaced the old inaccurate method of counting the number of cells to the field in a coverglass preparation made from a centrifugalized sediment. They first employed a modification of the Thoma Zeiss blood counting apparatus, as a more accurate means of estimating the number of cells. The technique employed by the writer was that described by Waterman in a recent publication ("The Value of Lumbar Puncture in Syphilitic and Parasyphilitic Diseases of the Nervous System"). The ordinary white blood corpuscle counter was used, drawing a staining mixture up to 0.5 and filling the tube to 11 with freshly-drawn cerebrospinal fluid which had been thoroughly shaken. The ordinary blood counting slide was employed, the cells within the squares on 10 slides being counted. This gives the number of cells per cubic millimeter. The staining mixture employed was methylene violet 0.1, acetic acid 2, and water 50, which mixture seems to bring out the character of the cells. The writer found that the staining mixture was not necessary, as good results can be obtained by substituting glacial acetic acid.

To quote Waterman: "The value of this method lies chiefly in its accuracy, allowing the results of one investigator to be compared with those of another, without question of any variation depending on technique. In this way it may be possible, by collecting a large number of cases, to make fine distinctions between a number of conditions in which lymphocytosis has been found, and we do not need to deal with such terms as "positive results," "moderate increase," and "marked increase."

All the evidence goes to show that the normal fluid, by this method of counting, demonstrated from no cells to three cells in a cubic millimeter. For the purpose of comparing the counts in tuberculous meningitis with normal and other diseased conditions, the writer has taken ten cells per cubic millimeter as the possible upper limit of the normal count, regarding the normal standard as from 0 to 10.

Of course the cell count is regularly increased in the other forms of meningitis, the increase being usually enormous, far above that of tuberculous meningitis, in early cases running into the thousands, but these cases are usually easily distinguished from tuberculous meningitis by means of other characteristics of the cerebrospinal fluid, such as the predominance of polynuclear

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cells and the presence of the specific microörganism involved. It has been shown also that there is nearly always a moderate or marked increase in the cell count in cases of (1) syphilis with active secondary lesions, (2) syphilis of the central nervous system, (3) syphilitic eye disturbance, (4) tabes, (5) general paresis. In addition to these an increase may be found in (1) multiple sclerosis, (2) mumps, (3) herpes zoster. Many of these conditions are rare in childhood, and none of them are conditions which are apt to be in question in considering the diagnosis of tuberculous meningitis.

The writer's records include 71 examinations, representing 60 cases, of the number of cells per cubic millimeter found in the cerebrospinal fluid of cases of tuberculous meningitis. The count was above the normal in all cases. The lowest count was 30, and the highest 920. These cell counts may be seen from the following table:—

TABLE II.

Cells per Cubic Millimeter.	Number of Counts.
0- 10	0
10- 50	5
50-100	12
100-200	25
200-400	25
over 400	4

This table shows the amount of increase above the normal usually found. From another point of view, the following table shows the percentage of cases in which the increase reached certain figures:—

TABLE III.

Cells per Cubic Millimeter.	Percentage of Cases.
Over normal	100 per cent.
" 50	93 " "
" 100	76 " "
" 200	40 " "
" 300	15 " "
" 400	5 " "

From these results the inference may be drawn that an increase in the number of cells per cubic millimeter found in the

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cerebrospinal fluid in tuberculous meningitis is constantly present and is usually considerable. There is, indeed, quite a wide margin, of 20 cells per cubic millimeter, between the upper normal limit and the lower limit found in tuberculous meningitis. This is diagnostic in distinguishing tuberculous meningitis from patients who are not ill.

In order to determine how far an increase in the cell count is diagnostic of tuberculous meningitis, we must know whether the cell count is increased in other diseased conditions, and, if so, what is the extent of the increase.

The writer has had the opportunity to perform lumbar puncture and to estimate the cell count of the fluid in a considerable number of other diseased conditions. This opportunity occurred in connection with work with the Flexner antiserum for epidemic meningitis carried out during the last two and a half years, in the course of which lumbar puncture was performed in every case in which meningitis could not be positively excluded. The results are of particular value, as they are based on a class of cases which did, and frequently do, bear a notable clinical resemblance to the various forms of meningitis. The results of this investigation may be seen in Table IV.:—

TABLE IV.

Disease.	Cases with Cell Count Normal.	Cases with Cell Count Increased.
Lobar-pneumonia	14	0
Anterior poliomyelitis	6	0
Typhoid fever	3	0
Solitary tubercle	2	0
Cerebellar tumor	2	0
Cerebral hemorrhage	2	0
Rheumatic fever	3	0
Otitis media	4	0
Measles with pneumonia	1	0
Gastroenteric intoxication	7	0
Tetanus	0	1

It appears from these results that no increase in the cell count above the normal occurred in any of the diseases most commonly mistaken for meningitis, the only case in which such increase was

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found being the case of tetanus, in which the cell count was 60. This case was not mistaken for meningitis, the lumbar puncture having been performed for the purpose of injecting magnesium sulphate into the spinal canal. Therefore, although the number of cases examined is still too small to permit the drawing of positive and final conclusions, yet there seems to be weighty evidence for regarding a marked lymphocytosis as a diagnostic sign of tuberculous meningitis, provided that the cell count is estimated by an accurate method. The presence of this definite and measurable lymphocytosis, if constant, is an important addition to our means of diagnosis. It is hoped that an accurate method of estimating the lymphocytosis will be generally adopted, particularly in cases coming to autopsy, in order that the value of this sign in diagnosis may be finally determined.

The Value of an Accurate Estimation of the Lymphocytosis in the Cerebrospinal Fluid.—This procedure has already proved of great value in many doubtful cases. The writer has records of cases in which the diagnosis was extremely doubtful, but in which a cell count of several hundred gave evidence of tuberculous meningitis, and in which this diagnosis was later confirmed by autopsy. On the other hand, the accurate estimate of the cell count has proved useful in other doubtful cases, in which a normal cell count gave evidence against tuberculous meningitis, and the subsequent developments proved that this evidence was correct. The procedure has been of especial value in the differential diagnosis between tuberculous meningitis and the cases of gastroenteric disturbance and toxic absorption which frequently simulate it. In a number of the latter cases tuberculous meningitis has been early excluded by the cell count, greatly to the relief of the parents, and in every such case in the writer's series the favorable prognosis has been justified by developments. The writer recalls one case in particular, in which a consultation of Doctors Rotch and Jacobi decided that tuberculous meningitis was the most probable diagnosis, but in which the cell count was normal. The case later proved to be one of intestinal autointoxication.

There appeared to be no definite relation between the stage of the disease and the amount of the lymphocytosis. A very important fact was, however, that the lymphocytosis was found in every case, no matter how early, the series including a number of cases examined on the second or third days of illness.

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With reference to the possibility of recovery from tuberculous meningitis, which was the original theme of this paper, the writer believes that if the cell count is proven to be a positive diagnostic sign there is every likelihood that the evidence in favor of such possibility will be multiplied, for if there is a possibility of recovery which has been prevented from standing out clearly by the difficulty of making a positive diagnosis, it should presently appear if the difficulty is lessened. The writer has records of 3 other cases of apparent recovery, in which a diagnosis of tuberculous meningitis was based on the clinical features, with a cerebrospinal fluid showing a high lymphocytosis as measured by the cell count, but in which tubercle bacilli were not demonstrated. These cases are not scientifically proven cases of tuberculous meningitis, and not worth reporting, unless the cell count is a positive diagnostic sign. If it is, they increase the evidence in favor of a possible favorable outcome in this fatal disease.

Early Lumbar Puncture Recommended.—In regard to the treatment of these cases, is there not a possibility that in the case reported above the very early performance of lumbar puncture might have had some favorable action? It is noteworthy that in 2 cases of recovery reported from Copenhagen, in which bacilli were found, it was stated that the treatment was by repeated lumbar puncture. In the writer's 3 unproven cases of recovery, in which the diagnosis was based on the cell count, it is noteworthy that in all three lumbar puncture was performed unusually early in the course of the disease. Most cases of tuberculous meningitis do not receive early lumbar puncture. The cases in the hospitals are usually seen late in the course of the disease. In private practice, and in many early hospital cases, the procedure of lumbar puncture is postponed until the gradual development of the disease has brought on unconsciousness. The writer is inclined to advocate, in suspected cases, early lumbar puncture, with an accurate estimation of the cell count.

Summary Conclusions.—In view of the above case, and the few other cases of recovery reported in the literature, and of the evidence on which the prevalent idea of the fatality of the disease is based, the writer believes the conclusion is justified that it is not proven that tuberculous meningitis is invariably fatal; that there is some evidence of the possibility of recovery; and that there is a possibility that this evidence may be increased by in-

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creasing the frequency of making a positive diagnosis through an accurate estimation of the cell count.

If these conclusions are justified it is of great importance to us in dealing with the parents and relatives of our patients; in the matter of prognosis, we need never give an absolutely fatal prognosis. The writer is accustomed in these cases, while not minimizing the seriousness of the condition, to give two faint rays of hope. The first is that his diagnosis is wrong, the second is that there is some evidence of the possibility of recovery.

DISCUSSION.

DR. NORTRUP.—I think Dr. Dunn has undertaken a very large proposition. If he is willing to go on record that this method of diagnosis is accurate I have nothing to say. I have had one of these "don't know" cases myself. I should like to ask Dr. Dunn to what age he limits this diagnostic method? The case I refer to had convulsions, persisting vomiting, retraction of the head, coma, Kernig's sign, stiff neck and opisthotonos, sighing respiration, etc. Lumbar puncture gave increased fluid and increased lymphocytes, but the patient suddenly began to get better; why, we do not know, and so I call that a "don't know" case. The only exact method, of course, would be the finding of bacilli.

DR. MEARA.—I have lately correlated the cases of meningitis of the last three years in Bellevue Hospital. Of 50, in which lumbar puncture was done, in 47 the bacilli were recovered. The technical work was done by Dr. Charles Norris. The results correspond very closely with those of Dr. Dunn. There was also in some cases an increase from .02 per cent. to .09 per cent. in proteid. One child under one year, in which the bacillus had been found in the fluid, so improved after some weeks that we thought we had a case of recovery, but the child suddenly got worse and died. The blood count in this series showed decided leukocytosis, some as high as 36,000. The polymorphonuclear count greatly increased; in some as high as 90 per cent. There were a number of cases of so-called meningismus in which there was slight increase in cells. I think Dr. Dunn said the frequency with which the bacilli were found in the fluid was not so great, but I think our methods were accurate.

DR. HAMILL.—I have not formally reported, but in discussion I have referred to the recovery of a case of tuberculous meningitis confirmed by the demonstration of tubercle bacilli in the cerebrospinal fluid, which was under my care a few years ago. I never laid much stress upon this case because no animal in-

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jections were made and so the final link in the chain of evidence was lacking. I never hesitated, however, to believe that it was a case of tuberculous meningitis and that the organism found was the tubercle bacillus.

DR. GRIFFITH.—As a matter of history, I would say that Fürbringer, who was the first to bring lumbar puncture into practical prominence, reports, as I remember, the finding of the tubercle bacillus in a case which afterward recovered.

DR. CHURCHILL.—The presence of the bacillus is the only evidence that can be considered positive. The increase of lymphocytes is not sufficient, though some of these cases which show lymphocytosis may be cases of meningitis.

DR. ABT.—I believe cases of tuberculous meningitis may recover. I remember as a student in Vienna Professor Goliski used to show the brain of a child in which the diagnosis had been made and in which the child recovered and the diagnosis was supposed to be doubtful. The child finally died of some other disease, and the old scars of the base of the brain were evident, and no doubt was left as to the question of its being a case of tubercular meningitis.

DR. DUNN.—I might say that in a part of the paper which I omitted reading I called attention to the diseases in which there is an increase of the lymphocytes, which included syphilis. The main point I wished to make was that the regular methods of demonstrating an increased number of leukocytes has been too inaccurate. I do not think the method of slide counting has been widely enough employed. A standard for normal fluids has been established. Waterman did a lot of work in that line. An increase of the lymphocytes has only been found in the diseases I mentioned in my paper. In none of my cases was there a count above 10 in meningismus.

Dr. Flexner said this morning that an increase of lymphocytes is present in early stages of poliomyelitis. As to the bearing of the number of leukocytes on the possibility of recovery, it may not increase the positive evidence, but it may be of great help if it proved that the increase in the number of lymphocytes will give us a diagnostic standard.

DR. ADAMS.—The writer made the statement that we should not be without hope in these cases; that we should advise parents to have a good deal of hope. That being the case, and granting that the diagnosis has been proven, upon what does he base his prognosis of probable cure?

DR. DUNN.—I do not like ever to give an unfavorable prognosis. I tell them that there are two possibilities of hope—one that the diagnosis may be wrong, and again that some cases, although very few, may recover.

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RETAINED INTUBATION TUBE (SECOND INTUBATION). DIPHTHERITIC PARALYSIS. RECOVERY.

BY W. P. NORTHRUP, M.D.,
New York.

Presbyterian Hospital service, ambulance admission, great urgency. W. K., fifteen months old, left Willard Parker Hospital four weeks before this history begins. He had been in that institution four weeks suffering from diphtheritic laryngeal stenosis and wearing an intubation tube two of those weeks. He had had whooping-cough four months before contracting diphtheria. Since leaving Willard Parker Hospital he had been well, except for a cough. Three days before entrance he began to be "croupy." This was four months after whooping-cough and two months after beginning diphtheria. He was better the day following, of croup, worse again the succeeding night and was "bad all night." The next day he had no relief. All day he grew worse, till late in the evening the mother thought he was about to choke to death; indeed, she thought him dead for a few moments.

He came into hospital in such urgent dyspnea that immediate intubation was attempted by the ambulance surgeon. Insertion of the tube was very difficult and at first attempt unsuccessful. However, the unsuccessful efforts at intubation resulted in temporary relief.

My first attempt at intubation was not successful. The tube met with obstinate resistance below the cricoid ring (subglottic edema or swelling). The very smallest tube was gently forced past the obstruction and relief afforded. So great was the dyspnea, so great the exhaustion from the repeated attempts to insert the tube, that artificial respiration was immediately necessary.

The first diagnosis was catarrhal laryngitis in a larynx recently injured by diphtheria and intubation, a marked nervous element being added from a recent whooping-cough. There was no pseudomembrane visible, tonsils were large, coarse râles were to be heard everywhere over both lungs, and the child was in a poor general condition.

Two days after admission respiration through the tube was free. Pulse, 180-190; temperature, 102,° 104,° 105.4° F., and for nine days thereafter the temperature ranged between 102°-

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103° F.; after that it sank to normal and so continued. During the elevation of temperature signs of consolidation appeared at one apex and the gloomy prognosis of "the usual" ending was entertained. No diphtheria bacilli found in throat cultures.

After three days the tube was taken out, we still believing it to be catarrhal and mainly spasmodic. To insure quiet and to allay spasm the child was placed in a far corner of the ward, face to the wall, attended by a nurse familiar to its sight, and in addition given a fair dose of paregoric and ipecac. After a brief sleep it awoke, seemed frightened, choked up and ceased to breathe. With the greatest difficulty the tube was reinserted, artificial respiration was performed and the child given one more chance for life. If it had not been surrounded by all the necessities and even the luxuries of emergency resources it would have died then and there. In fact, this quick emergency relief was required a half dozen times in the next few days.

During the first month in hospital he proceeded to cough out all the tubes that could be put into him. Sometimes he would cough it out before the doctor could get out of the ward. More commonly he coughed it out just after the doctor had comfortably dozed off into his interrupted sleep again. It is estimated that he required intubation forty times during his stay in the hospital. Ever increasing sizes of intubation tubes were required until this fifteen-months child was requiring a tube proper for a five-year-old child. After four weeks in hospital he was able to go twenty-four hours without the tube for the first time. Two weeks later he again went twenty-four hours without it. Just seven weeks after entrance he went without the tube six and one-half days, again requiring it. In the effort to quiet spasm, for that seemed the greater part of the difficulty, he was given paregoric, ipecac and, what was a potent element, he was petted and soothed to do away with panic. He was frightened. For days and nights the nurses, and often the doctors, carried the little patient on their shoulders, his nose buried in their necks. Everybody became interested in his recovery and willing to do anything to further it. I mention this as an important and successful part of the treatment, and an element which was often mentioned by O'Dwyer. This was practically the last needed intubation. Duration of necessary intubation, seven weeks.

Five weeks after admission the temperature rose to 103.5°F., 102°F., 101°F., etc. This time it was presumed that he had pneu-

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monia and perhaps that was the kindest thing that could happen to him. Our minds were filled with remembrances of past experience and the thought that he was to wear the tube for an indefinite period and that sooner or later he would require an operation on the larynx, after which he would succumb to pneumonia.

Fortunately, he now entered upon an unbroken convalescence, but with ever threatening recurrence of stridulous respiration and nervous dyspnea, many times narrowly escaping reintubation.

The patient was discharged from hospital, and later returned by the mother, claiming that he was very dyspneic again. It is not sure that she did not wish him to again profit by the advantages of good food and care and wished to board him with us. However, the little patient had been the subject of so much care and of so much solicitude that he had won his way to the hearts of those in the care of the ward and was kept willingly. He is there to-day. The nervous element is even yet very pronounced. The least excitement puts him into stridulous respiration, till he can be heard about the ward plainly. Even the excitement of his tray of food coming into the ward starts him into stridor. He has afforded us ample opportunity to study his case fully. Among other things, his voice is coming to him. He is not old enough to talk, but he is able to phonate, speaking a word or two. His pharynx looks healthy; we can see the epiglottis and top of his larynx, but not the cords. The case is now complete. We believe him to be well.

Résumé.—When thirteen and one-half months old patient wore an intubation tube in Willard Parker Hospital for laryngeal diphtheria. Recovered and was discharged "cured." Was at home four weeks, came to Presbyterian Hospital when fifteen months old in urgent dyspnea, reintubated repeatedly with ever-increasing sizes of tubes for seven weeks; complete recovery. Secondary dyspnea would seem to have been due to diphtheritic laryngeal paralysis, plus subglottic swelling, also complicated with a marked nervous spasm, the tendency to which was largely contributed by his previous whooping-cough. It is hardly proper to call this retained intubation tube. It is rather reintubation for late paralysis. The difficulty in settling the tube into place in the first intubation was due to subglottic swelling; the ever-increasing size of required tube was due to adductor paralysis; the inability to breathe when the tube was out was due to abductor

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paralysis. When the paralysis passed away in the course of time the functions of the intrinsic laryngeal muscles were restored and the patient recovered naturally.

In volume IX. of this society may be found O'Dwyer's last communication before this gathering, entitled "Retained Intubation Tubes." He says: "The cause of persistent stenosis following intubation in laryngeal diphtheria can be summed up in the single word traumatism. Paralysis of the vocal cords may possibly furnish an occasional exception to this rule." Again, "Paralysis of the vocal cords as a cause of retained intubation tubes I confess I know little about." Also, "Paralysis of the intrinsic muscles of the larynx is acknowledged to be a rare sequel of diphtheria. I have never observed it." O'Dwyer, later in his paper, uses the expression: "In adductor paralysis, in which the retaining power of the vocal cords is lost," etc. Obviously O'Dwyer thought only traumatism was to be blamed and that due to faulty tubes and faulty methods of intubation. The present case is unique in my experience and reading.

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CHRONIC RHEUMATOID ARTHRITIS OF CHILDHOOD.

BY HENRY KOPLIK, M.D.,
New York.

Rheumatoid affections of the joints in children have of late years attracted unusual attention both in England and America. The cause of this study lies rather in the obscurity of the nature of many of these affections than in their common occurrence. The term rheumatoid is not such a very apt one, as it is intended to include certain varieties of joint affections to the exclusion of others. In this paper I shall exclude from consideration all forms of joint affections which ordinarily complicate the infectious diseases or gonorrhea. Nor do I include in my study any form of joint affection which presupposes changes in the bony structures, making up the joint, as seen in syphilis or tuberculosis. There are two forms of joint affection which have lately received considerable notice, the one described by me in an article in the ARCHIVES OF PEDIATRICS under the caption of arthritis deformans, that described a short time after by Still as rheumatoid arthritis, and a third form also hinted at by Still and others, a form of joint affection closely resembling articular rheumatism, in which the swelling of the joints is rather of a subacute nature than chronic; a number of fibrous nodules occur on the tendons in the vicinity of the joints, and there is an enlargement of the lymph nodes throughout the body. In all of these forms of arthritis, "rheumatoid," if they must so be called, the course of the disease resembles very much a form of infection, though, after the onset of the joint trouble the evidences of infection, such as fever, are apt to subside and the joint swellings and other manifestations of an infection of some kind continue. Charcot, Weil, Osler, Henoch, Barlow, Pasteur have described forms of rheumatoid arthritis. The form described by Charcot and Weil and subsequently by Osler, is extremely rare and Henoch so classes this form, which closely resembles that seen in the adult, as arthritis deformans. In children the form described by the writers men-

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tioned as arthritis deformans begins in children after some exposure in a distinct infective way, and it is more than likely that it has very little relationship to the adult form, for at first in children the joints and periarticular tissues are involved, and it is only very late, if at all, that the bones and muscular tissues become affected. I think that in children what has been described as arthritis deformans so closely resembles another form, the so-called Still's form of rheumatoid arthritis, that they both are probably the same form of disease and are peculiar to children alone. I have been led to this conclusion by a study of quite a number of these cases of rheumatoid affection of the joints.

In 1896 I published a case of rheumatoid arthritis, the history of which I append. This case so closely resembles the cases which were subsequently described by Still, that to make them two separate affections would only confuse matters by an unnecessary refinement of classification.

CASE I. Girl, *aet.* six and one-half years, June 11, 1894.

Family History.—Father not well(mother did not know what the trouble was); mother well. Four children living. One child died, aged two years, of meningitis. Mother had one miscarriage at four months. The other children are well; the mother is not rheumatic, neither is the father. They have never lived in a basement and now live on the third floor.

Patient's History.—Labor prolonged; was a healthy breast-fed baby; nursed one and one-half years; first tooth at one year; walked at one and one-half years; talked about the same time. When three years old she had measles, and about sixteen months ago pneumonia, with which she was ill two weeks. She had quite recovered when present illness began.

Present Illness.—About a year ago the child was taken sick with fever; this alternated with chills and lasted two weeks. During this time she complained of severe pains in the feet. The mother says that she could ascribe no cause for the illness. The child had not been put out in the rain nor had she had damp feet. She had been sleeping on the roof during the hot nights, but had done it some time before the illness began. After the chills and fever left her the pain and soreness in her legs and feet remained. She walked still, but very slowly; she returned to school for a few days, but walking was so painful she was obliged to stop. The pain in her feet seemed to center about the ankle, which became swollen; the pain in about a month seemed to go to the

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hands and wrists. They began to swell slowly; there was still pain in the feet and ankles, but not as sharp as in the hands. This had been going on several weeks, when her knees became painful and they too grew larger. The pain had always been worse at night, and would frequently waken her from a sound sleep; when quite bad she could not get to sleep again, but at other times would start up with a sharp cry several times in the night. After she had been ill two months she could not walk at all. When sick three months she was taken to the hospital, where she remained seven months. While there she contracted urogenital blenorrhea, which lasted some weeks. While there the articulation of the head with atlas and axis became involved and the pain in the neck and stiffness began. The elbow joint likewise became involved. The mother said she was not certain, but thought the elbow joint was not involved at the time the child was taken to the hospital. She has been home from the hospital two months and has grown steadily worse.

Status Praes.—The child always has some pain. It varies in locality, being present sometimes in one joint, sometimes in another. She has pain when she is moved or lifted. The pain has been very bad during the last week and seems to affect especially the occipitoatloid joint. Sometimes for several hours she cannot move the head at all without pain.

Position of child is as follows: The head is constantly in a semiflexed position; the forearms are semiflexed; extension draws on the muscles of the arms and gives a strained feeling. The legs are in attitude of semiflexion, the feet being slightly extended. The child cannot lie down, but takes the semi-recumbent position. She is very pale. The head is small; the chin pointed; the conjunctival mucous membrane is pale; the tongue is coated with thick gray coat. The teeth are twenty-two in number, the tonsils are normal. The child says that opening the mouth wide gives pain in the back of the neck, not in the maxillary articulation.

When told to rotate the head she can rotate an arc of about 20° to the right; not so far to the left; extension of the head is impossible.

The neck is normal; chest well shaped; the lungs normal. *Heart.*—Second sound at apex somewhat accentuated. *Abdomen.*—Very tympanitic. *Liver and Spleen.*—Not felt. *Articulations.*—The maxillary joint is free. At the shoulder joint the measure-

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ment of two sides is the same. There is no apparent enlargement.

Movement.—The child can raise both arms above head without pain.

Elbow Joints.—Both are enlarged, palpation reveals fluctuation and also enlargement of ends of the bones. The bone enlargement begins about 5 c.m. below the joint, with a gradual upward thickening. Flexion of the forearm on the arm is good; extension is very much limited; backward movement of the arm gives pain and is very limited.

Wrist Joint.—Fluctuation is felt; the dorsal enlargement of carpal bones toward the radial side is marked, and is the shape of a hemisphere, the enlargement being more marked on the left side; flexion of hand is perfect; epiphyses of the metacarpo-phalangeal articulations are enlarged. Flexion is perfect; rotation very much diminished; extension the same.

Hip Joint.—There has never been any pain *in* the joint, but cannot get movements, as it gives pain in muscles of the thigh on account of knee-joint deformity.

Knee Joint.—There is a globular enlargement of the knee joints, slightly larger on left side, which measures 1½ cm. more than the right. She can flex the leg on thigh, but that is the only movement possible.

Ankle Joints.—Both present diffuse enlargement, the left measuring 21 c.m., the right 19 c.m. The foot is held very stiff, adduction and abduction of foot being very slight. Movement of the toes is slight. The epiphyses of phalanges of toes enlarged.

There are no glandular enlargements. The bowels are constipated; the appetite is very poor. The respiration is 24; the pulse 96; the temperature 98°. There is slight vaginal discharge. Cover-slips show gonococci.

November 1, 1895, the movements described above have become more and more limited. Even flexion, which was present at first, is limited. The child sits and sleeps in a fixed upright attitude.

We do not lay much stress on the vaginal discharge as suggesting gonorrheal joint trouble, because the disease had practically disabled the child when she was admitted to the hospital, where she contracted the vaginal disease.

Discussion—In this case the onset was acute in the manner of an acute infection with fever, accompanied by pain in the joints

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with subsequent swelling. The joints, beginning with the ankles and larger joints, slowly became involved one after the other until the child was practically disabled. The cervical vertebræ became affected, thus demonstrating the general infective nature of the disease. Finally, the smaller joints became the seat of disease. In this case, coming under observation fully a year after the onset of the affection, the lymph nodes, spleen and liver were not enlarged. It will be shown in the cases that are to follow that the enlargement of lymph nodes, as also the spleen and liver, is seen mostly at the outset; that subsequently they may attain the normal size, or, for the most part, retrograde to the normal.

In 1897 Still described a number of cases of rheumatoid arthritis, so-called, in which most of the cases, fully half, were affected before the second dentition with fusiform enlargement of the joints. There was no redness, but the glands, or lymph nodes, in the axillary, epitrochlear and inguinal regions were enlarged. In some the spleen, in others the liver, was enlarged. Three autopsies in such cases demonstrated adherent pericardium in one case, in another valvular involvement.

Similar cases have been described by Chaufard and Raymond, also by Ballantyne and Wohlman; and in America, in 1899, by Taylor and McKim. In order to complete literary mention cases have been published by Hunt, Poynton, Mouriquand and Edsall.

The affection described by Still certainly resembles in its onset an infectious disease. It differs from the rheumatic attack by a chronic progressive involvement of all the joints, beginning with the larger and taking in the smaller joints later on, with a tendency to avoid the cardiac structures.

CASE II. The following case is illustrative of this form of arthritis:—

J. N., male, three years of age, Russian parentage. Was admitted to the hospital October 13, 1908, with a family history, which was negative. The patient had never had any exanthemata, presented no hereditary history, was healthy up to the present illness.

The present history is of ten months' duration, began gradually with pain and redness and swelling of the right ankle joint. This lasted for several weeks and then subsided, so that the child could walk and play. Since then there has been recurrences of

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swelling of the right ankle, which is weak, combined with painful swelling of most of the large joints.

During the past five months the swelling of the joints has persisted; there was occasional fever, especially toward evening, accompanied by profuse sweating at night; there was no nausea or vomiting; the swelling of the joints confined the patient to bed.

During the past six months the mother noticed that swellings appeared on the neck and groin; about the elbow these swellings were painful. The child developed stiffness of the neck, combined with pain in the spine, dorsal and lumbar regions. He has emaciated considerably, his appetite is poor, the bowels are constipated, acting only with cathartics. The chief complaint, therefore, is pain, swelling of the joints, fever accompanied by sweating and emaciation; inability to move the joints or to walk; pain in the neck, dorsal and lumbar regions; swelling of the glands in the neck and groin.

On admission to the hospital the house physician's examination showed the patient to be in poor condition, pale, weak, emaciated. The head, eyes, ears, nose, mouth and teeth presented nothing abnormal. The fauces showed hypertrophy of the tonsils, otherwise negative. Larynx, trachea and thyroid were normal. Lymph nodes, posterior and anterior cervical were enlarged in packets. The supraclavicular and axillary lymph nodes very much enlarged in packets; epitrochlear in size of a large lima bean, in groups of some size, and in axillary regions the inguinal lymph nodes were very much enlarged; the lungs presented dullness, especially behind and on both sides, toward the base of the lung; diminished voice, fremitus, but no râles; the heart presented nothing abnormal, no murmur; the liver was palpable a finger's breadth below the ribs; the spleen was not palpable on account of tympanites, the abdomen was large and distended, presented nothing abnormal. There was bilateral cryptorchidism; the extremities showed marked atrophy of the muscles of the arm, forearm, thighs. Elbow joint showed a deformity, square in character, which shall be described; no limitation of motion and no crepitation; the wrist joints were swollen with a gelatinous effusion; the finger joints were not deformed; and in joints which were swollen there was no effusion; no crepitation; some fluid in the right knee; both ankle joints were uniformly swollen and effusion was present, and some considerable swelling around the joint.

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The entire spine was held rigid and was very tender at time of admission. A tuberculin cutaneous test was negative. The red blood cells were 4,600,000; white cells, 29,000, of which 78 per cent. were polynuclear cells.

In consideration of the condition of the joints I made the following examination, and found the joints of the elbows, wrists, knee and ankles considerably swollen. The elbow on the right side showed a distinct sac of effusion; on each side of the olecranon process and on the anterior aspect of the joint there was an apparent distribution of fluid and a distinct fluctuation and a gelatinous sensation to the effusion. The extremities of the bones were prominent, but this was probably on account of the great emaciation. The right elbow presented very much the same appearance of effusion into the joint; anteriorly and to the left, and the epiphyses of the bone entering into the formation of the joints on both sides, appeared larger than normal, as the subsequent history showed, on account of emaciation, but were not really enlarged.

On the palmar aspect of the hand the effusion seemed to be mostly around the situation of the pisiform bone, extending down the palm of the hand; the lower end of the radius was not thickened; the tissues around the metatarsal bone of the dorsal aspect of the hand were slightly thickened; the bones and joints of the fingers were apparently normal; the knees on both sides were enlarged, and there is effusion above and below the patellar joint; there seems to be a little fluctuation behind.

Over the popliteal space the extremities of the bones entering into the joint were only apparently enlarged on account of the emaciation.

Both ankle joints were enlarged; fusiform in shape, with evidences of fluid behind and below and on both sides. The tissues on the dorsum of both feet seem thickened; the metatarsal and phalangeal joints were normal. The patient held the head rigid; the head drooped slightly forward; there were enlarged glands on both sides on anterior border of the trachea extending down to the clavicle the size of a large white bean; over the situation of the third dorsal vertebræ there was a distinct swelling about the size of a pigeon's egg, painful to the touch; over the seventh dorsal there was another swelling; at the dorsal lumbar junction there is a slightly angular deformity. The whole spine is exceedingly painful; over all the joints, with the exception of the spine,

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there is very little pain; no limitation of motion, no crepitation, as though the bones or cartilages were eroded or exposed. The heart is normal.

Both wrist joints were aspirated, but nothing obtained. The needle entered into the tissues rather around the joint than into the joint itself; in other words, the joint cavity was not continuous with the swelling over the joint.

The second cutaneous tuberculin test was negative, as also the third.

October 25th. The condition is good; there is some improvement in the condition of the joints; the lymph nodes have diminished in size, especially the epitrochlears. The spine is still tender and still presents the swellings in the lumbar region, but they have apparently subsided in the dorsal region and some in the lumbar region.

The leukocyte count at this time gives: white blood cells, 25,300; polynuclears, 66 per cent.; lymphocytes, 24 per cent.; large lymphocytes, 6 per cent.; eosinophiles, 3 per cent.; basophiles, 1 per cent. Some occult blood in the stools by the guiac test.

October 30th. The patient's general condition has improved, but the child resents any interference as far as the neck is concerned; head, too, is rigid; patient does not move the spine; some blood is passed in the movements. There is an iodid of potassium eruption on the face; the joint swellings are gradually becoming smaller. The Wassermann test proved negative, performed by Dr. Noguchi.

November 5th. Patient has steadily improved, but only up to a certain point. The swelling of the left wrist is still present to a marked degree; the tissues about the right wrist are still swollen the same as on admission; the elbow joints are markedly improved, so also the spine; the lumbar swelling has disappeared; the spine is held rigid; the ankle joints are still puffy and swollen, although not nearly so much as on admission; no pain in these joints.

The patient refuses to use the lower extremities, possibly on account of the pain.

The condition on discharge showed all joints practically in a normal condition, with the exception of a slight puffiness and increase of size in the ankle joint; the neck was still slightly rigid, but patient moved without any pain. The spine was normal,

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glands have been reduced to an extent inappreciable to what they were on admission; absent in the axillary glands, epitrochlear region, neck and groin.

Patient was walking about the ward, increasing in weight and quite happy.

The temperature is worthy of note in this case. On admission the temperature ranged from 101° to 104° F., with remissions to the normal daily. After a week the temperature subsided so that it only reached 102°F. occasionally at intervals of three days, and then gradually subsided to the normal. The leukocytosis is worthy of note.

Discussion.—After the second week of the stay in the hospital the temperature fell and remained at the normal.

During the latter period of the child's stay in the hospital there were occasionally rises of temperature to 101°F., with enlargement of the glands connected with the tonsils, and there was always present a slight hyperplasia of tonsillar, nasal, pharyngeal and adenoid tissues.

Another point of interest is that the leukocyte count up to November 9th remained high—20,000 on a slight rise of temperature; would fall, however, when the temperature subsided, but never during the first, second, third or fourth weeks of the illness did it remain permanently low. The polynuclear count was high also; incidentally it may be mentioned that a blood culture proved negative.

That this case bears all the marks of an infection of some kind no one will gainsay. The mode of onset, the temperature, and remissions even after months of illness, the high leukocyte count, the subsidence of the symptoms with retrogression of glandular enlargements with final evident improvement of the patient are extremely interesting, in view of the gloomy prognosis in most of the cases published by Still. The absence of any effusion in the cavity of the joint, the negative blood culture and the negative of syphilis by the Noguchi-Wassermann test are well worth more than passing notice. In this case there was some blood in the stools by guaiac test. In the case which follows there is also a history of an intestinal hemorrhage on admission to the hospital. This was not repeated.

CASE III. Male, eight years of age.

Family History.—Negative—not obtainable.

Previous History.—Measles, complicated by pneumonia six

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years ago, after which hands and feet were swollen and painful for one year. Since then child has had recurrent attacks of pain and swelling of hands and feet.

Present History.—For the past eight days there has been, in addition to an exaccerbation of the painful swelling of the hands and feet, swelling of the abdomen, painful defecation with passage of small quantities of blood in the stool, cardiac palpatation, dyspnea on exertion, weakness and slight fever.

RHEUMATOID ARTHRITIS IN A BOY EIGHT YEARS OF AGE.
Large and small joints affected; enlarged lymph nodes in large pockets; liver and spleen enlarged.

Physical Examination.—Child is pale, complexion pasty. Over the pelvis are several hemorrhagic spots; in the lips, herpes. There is a slight internal strabismus of right eye. Head is held in a rigid position, bent slightly forward, and any attempt to over-extend the head is resisted. Lateral motion of the head is unimpeded; normal flexion of head on chest. Over left frontal

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region is a small tender prominence, probably traumatic. The child complains of pain in the tempero-maxillary region when the mouth is opened wide. The axillary lymph nodes are the size of large beans, hard, discrete, not tender. The cervical, submaxillary, right epitrochlear and inguinal nodes are also hard and enlarged. The sternum is markedly prominent; there is bulging of the lower portions of chest. Marked tenderness over ribs and intercostal spaces of right lower chest. No sternal tenderness. Lungs show slight dullness at both bases more on the right side, where there are a few crepitant râles. The heart is normal, except for an accentuation of the second pulmonic sound. The liver extends down to the level of the iliac crest; its surface somewhat roughened, its edge firm and extremely tender. The spleen is felt just below the costal margin; it is firm and slightly tender. The abdomen is generally tender and distended; circumference at umbilicus, 58 cm. There is movable dullness in the flanks.

The Extremities.—Hands show marked thickening of interphalangeal joints, particularly at the proximal joints, giving the finger a peg-shape form. The terminal phalanges are flexed. The joints are tender, but not red. There is marked periarticular thickening and distinct crepitus at the right wrist; thickening at the left but no tenderness or crepitus. *Right elbow* is held semiflexed; it is too tender for examination. *Left elbow* also held flexed. About the joint is a tender thickening.

Knees.—Swollen, very tender. Circumference just above left patella—25 cm.; above right, 34½.

Feet and Legs.—Edematous. First left metatarso, phalangeal articulation swollen, red and tender.

Von Pirquet Test.—Negative (right and left arms).

Blood Count:—

Red blood cells	3,500,000	
Hemoglobin	68	per cent.
White blood cells	12,000	
Polynuclear cells	63	" "
Large lymphocytes	7	" "
Small "	9	" "
Eosinophiles	1	" "

Urine.—Heavy trace of albumin; hyalin and granular casts.

Wassermann Test.—Complement fixation test—negative (Dr. Kaliski).

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Stools.—Negative for blood, ova and amebæ, but during the physical examination the child passed a small blood clot by rectum.

Course.—Temperature normal on admission. Five days later it rose to 102°-104° F.; a diffuse macular eruption appeared on the trunk, front and back; the child complained of more pain in the joints, and the blood count showed white blood cells 39,000, with polynuclears 88 per cent. The temperature fell after two days, but rose occasionally to 101°F. for several weeks. Thereafter, until discharged from the hospital, the temperature remained between 98° and 100° F. By July 19th the joint pains had subsided, the neck was less rigid, and the lung signs had disappeared. The liver still remained as large as on admission. Two weeks later the child was able to walk about.

On October 26th I found the following: Effusion in both knee joints; the left knee cannot be flexed. Of the toes the right great toe is the only one affected. Atrophy of the muscles of the dorsum of the foot, and of all the muscles of thigh and leg. All the finger joints are swollen and deformed. The muscles of the hand are atrophied; also those of the forearm. There is effusion in both wrist joints. The motion in both elbows is impaired. The neck is still rigid. Liver felt 10:5 c.m. below the costal border in the mammary line; it seems to be divided off into lobes. The heart sounds are weak, but otherwise normal; no signs of adherent pericarditis. Of the lymph nodes those in the right axilla are no longer palpable, but there is still enlargement of the left axillary, of the inguinal, the epitrochlear, the submaxillary, and those in the "bicipital groove." The child has gained in weight; the face color is good. There has been no blood in the stools.

This condition was maintained until discharge, January 2, 1910. The urine continued to show some albumin and casts, both hyalin and granular. There was no further gain in weight, nor did the joint conditions improve further. Even when discharged the child was practically unable to help himself.

The treatment consisted in the administration of potassium iodid in small doses, occasionally of salicylates, of mercury by inunctions, hot baths and local applications of heat and cold to the affected joints.

Discussion.—The onset of the disease occurred after an attack of measles. It was of six years' duration, with exacerba-

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tions. First the large joints and then the smaller ones became involved, and then we have the typical picture of swollen, chronically enlarged joints, rigid neck, large lymph nodes, liver and spleen, with exacerbations of the symptoms from time to time. In the intervals the leukocyte count approached the normal, and when there were exacerbations of joint pains with rises of temperature the blood count assumed the picture of a leukocytosis of the polynuclear type. In this case there was not only a history of intestinal hemorrhage, but also of a petechial and erythematous skin eruption. The patient improved up to a certain point and then the improvement ceased, the symptoms remaining stationary. Tests for the presence of tuberculous and syphilitic element remained negative.

CASE IV. Female, twenty months old; breast fed. Parental history negative. None of the exanthemata. About three weeks before her visit the mother noticed swelling of the right ankle and a day ago the left ankle, the second phalangeal joint of the middle finger of both hands. Some fever.

Examination.—No vaginal discharge. There is a soggy swelling of both ankles, with considerable bogginess of the periarticular tissues; there is pain on pressure. The joints above are enlarged in a fusiform swelling. The head is held rigid. The inguinal glands are enlarged to the size of large beans. No axillary glands enlarged, no enlarged liver or spleen, no cardiac lesion, no fever. The parents of the child could give no definite history of onset. The whole picture was that of rheumatoid arthritis of the type under consideration. The disease in its course was slow and sluggish. The joints remained large and but slowly grew small, and only after four months was the child allowed to stand on the extremities which, with massage, improved as to the enlargement, but after four months they were still not entirely normal, though free from pain. The acute onset, sluggish course, involvement of the large, then the smaller, joints, the enlarged lymph nodes, the rigidity of the neck, all speak for the identity of the affection in the face of negative results of tuberculin tests as with the two previous cases.

In this connection it is of great interest to note that there are all grades of rheumatoid affection, from the mild attack to the fully developed swelling of the joints described above. An example of a so-called abortive form of this infectious rheumatoid

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arthritis is the following case, which came under my notice recently.

CASE V. Male, three years old, native of the United States; was the first born; father and mother were healthy, the birth was normal and he was breast fed. According to the physician the patient had rheumatism (?) six months before he was seen. After an ordinary vaccination the right large toe became swollen, then the corresponding toe in the other extremity, then the wrist on the right side, then there was swelling of the right foot, so that the patient could not walk. At times, as on the day when seen, the shoulder joint was very painful and could not be touched, and was slightly swollen. Strange to say, when the patient was seen he was in excellent physical condition; weighed 34 pounds. He had enlarged tonsils and possibly adenoids, the spleen was large and palpable below the border of the ribs; patient was pale, but there was no enlarged joints, except a distinct puffiness over the dorsum of the right foot, a little puffiness of the right ankle joints; the glands of the groin were enlarged, as also in the axilla; there was no heart affection.

In glands of axilla, groin and neck there was some swelling, and some stiffness in the joints of the neck. There was no cardiac disease. There was no fever.

In view of the history of distinct infection after time of vaccination—preceding the swelling and painfulness of the joints, with the glandular enlargement and without any characteristic picture of acute articular rheumatism, and apparently evanescent character of the swellings, few in joints, especially as none were present on the occasion of the visit, and in the presence of a very large spleen and enlarged lymphatic nodes all over the body—I surmised that we might be dealing with a case of an abortive infection, rheumatoid in nature similar to that described, when fully developed.

I put the patient under treatment, sent him back to his physician, and when seen a month subsequently the condition was as follows:—

The glands, which had been distinctly full and of large size in the axilla, groin, epitrochlears and neck, had disappeared; the spleen also had diminished in size; the stiffness in the neck had disappeared. The mother stated that there had been an occasional reappearance of the puffiness over the metatarsal joints of

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the right wrist, but there was no swelling on examination at the time of the visit.

In other words, the patient seen a month before had only had two attacks of pain; his enlarged glands had disappeared; his color had returned, and he was apparently very much improved.

This would appear to prove conclusively that there are certain infections which cause a rheumatoid affection of the joints, combined with enlargement of lymph nodes and spleen, but that this infection may advance to full development of the picture described or retrograde without further serious manifestations.

That in the cases which I have described we have an infectious disease cannot be doubted, from the mode of onset, the enlargement of the spleen, liver, lymph nodes, in some cases intestinal and skin hemorrhages, the fever and the leukocytic blood picture. The slow recovery from the primary infection, with the recurrences of pain in the joints and febrile periods. The emaciation and anemia are also characteristic. Some cases which I have seen, notably that published by Manges, showed, among other things, a mild exophthalmus. The enlargement of the lymph nodes are not simple enlargement to little above the normal size, but these nodes are much larger than those seen in the adult forms of rheumatoid arthritis. The lymph nodes return to the normal size after a time and leave but slight indication of their enlargement. In my cases also the spleen retrograded to the normal, but in one case the liver remained enlarged.

I have recently seen cases of acute articular rheumatism affecting the ankles of both extremities in which the lymph nodes in the groin were greatly enlarged, but in which the remaining lymph nodes and spleen were normal in size.

There is another form of rheumatoid arthritis which occurs in children and which is exceedingly rare. It should be remembered that in this country rheumatism with rheumatic fibrous nodules is a rare disease, whereas in England it is quite common. I have recently seen such a case, in which the joints, though large and painful, were never enlarged as in acute articular rheumatism; the lymph nodes were enlarged throughout the body, but not to the extent seen in the cases described in this paper. There was no leukocytosis and little or no temperature. Scattered over the body, in connection with the fasciæ and tendons, were fibrous nodules, which disappeared after a time, and there was a cardiac

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lesion and some choreatic movements of the extremities. This case was as follows:—

CASE VI. Male, nine years of age. Family history negative as to tuberculosis and rheumatism.

Past History.—Measles and pneumonia at one year. Pertussis at three years of age. No other illnesses. No previous choreic or rheumatic manifestations.

Physically and mentally well developed. Appetite and bowels good.

Present Illness.—Began gradually six weeks ago, after unusual walking exertion, when child complained of fatigue and pain in joints. Continued at school for ten days, complaining of tiredness. Feet or face never swollen. No cardiac palpitation. Three weeks ago mother noticed choreiform twitchings of face, shoulders and hands. No fever, no vomiting. Child sleeps restlessly during the night, waking frequently and suddenly out of sleep and dreaming. About three weeks ago noted stiffness of hands and inability to dress himself on account of pain in the hands. Nodules on the shoulders noticed four days before admission.

The chief complaints were fatigue, choreiform movements and restless sleeping.

Physical Examination.—General condition, fair. Some puffiness of the face. Mouth, no Hutchison teeth. Teeth and gums, fair. Tongue, slightly choreiform movements. Pallor of mucous membrane. Throat, negative. Speech not affected.

Nodes.—Anterior and posterior cervical, moderately enlarged. Submaxillary on both sides, moderately enlarged. Epitrochlear, on both sides moderately enlarged. A few small glands along brachial vessels on either side. In both axilla on both sides. Small inguinal on both sides.

Chest.—Fairly well formed. Expansion equal. Lungs, slight dullness in left suprascapular fossa and left apex posteriorly. Otherwise negative.

Heart.—Upper border second rib. Right border a finger breadth from right border of sternum. Left border a finger breadth inside anterior axillary line. Apex fifth space, 1½ finger breadths inside anterior axillary line. Action.—Regular, rapid, fair force; soft-blowing systolic replacing first sound at apex,

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transmitted to axilla, heard over entire precordium. Accentuated second pulmonic sound. Distension and pulsation of veins of neck. Pulses equal, regular, rapid, fair force.

Abdomen.—Lax, tympanitic; no masses palpable. Liver, fourth space to free border; not felt. Spleen, not felt.

Extremities.—No edema. Knee-jerks present.

Genitals.—Negative.

Rectal.—Not made.

Skeleton.—Nothing felt in bones of face. Cranium shows two small nodular exostoses on each side of occiput.

Spine.—Excessive prominence of spines of first and second lumbar vertebræ. Tender nodules in midline on sacrum. On each posterior superior spine of ilium are nodules, causing an irregularity of normal contour. Nodular deposits over spines of first and second lumbar vertebræ.

Clavicles.—Sternum; ribs, negative.

Scapulæ.—Four small nodular deposits along spine of the right scapula, not bony in nature, apparently adherent to the bone. Two similar deposits on spine of left scapula. Slight bony prominence over each angle over which is a fibrous deposit apparently adherent to the bone.

Humerus and Radius.—Exaggeration of normal prominences. Slight nodular deposit over right external condyle and over capitellum of right radius. Nodules over metaphalangeal joints of all fingers in both hands.

Patellæ.—Marked prominence on inner border of each patella.

Femur.—Marked prominences on condyles of both femurs.

Feet.—Proximal end of metatarsal bone prominent. Subcutaneous nodule on dorsum of right foot, apparently attached to the tendon.

No rigidity of the spine. No crepitus or limited motion in shoulder, wrist or elbow. Apparent atrophy of extensors of both forearms. Atrophy of thenar eminence on both sides. Incurving of distal phalanx on fifth finger in each hand. Flexion contracture involving middle finger on each hand and index of right hand, preventing full extension. Some tenderness and ulnar deviation of all fingers at metacarpophalangeal joints. No limitation of motion or crepitus in hips, knees or ankles. No atrophy of lower extremities.

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Synopsis:
1. Pallor.
2. General glandular enlargement.
3. Cardiac lesion.
4. Nodules on normal bony prominences.
5. Nodules apparently connected with bursal periosteum and tendon sheaths.
6. Atrophy of extensors in forearms with condition in hands.
7. Puffiness of face.

In explanation of glandular enlargement: Those of neck and submaxillary region size of one-half white bean. Axillæ, one or two felt the size of a large pea. Groin, on both sides size of one-half white bean. Epitrochlear, size of pea, only two or three felt. In other words, glands or nodes palpable, but not like those in Cases II. and III.

November 23d:—

Hemoglobin	90 per cent.
White blood cells	10,500
Polynuclear	65 per cent.
Large lymphocytes	6 " "
Small "	29 " "
	100 per cent.

December 13th: Epitrochlear nodes still enlarged. Most of the exostoses found at first examination have since disappeared on hands and feet, with exception of bony exostoses and of occasional fibrous nodules—the nodules have disappeared. Cardiac condition as on admission.

December 30th: Good. All subcutaneous and periosteal nodules have disappeared. Lungs clear. *Heart.*—Presystolic and systolic murmurs. Action, regular, fair force, rapid. Five days ago patient had fever for two days, with joint tenderness and pain and urticarial eruption.

November 25, 1909: *One radiographic plate* of both hands shows a haziness in the contour of the heads of the third and

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fourth basal phalanges of the left hand and of the 3d basal phalanx of the right hand. The same is true of the bases of the corresponding middle phalanges. But this appearance may be due to position, the boy not being able to extend this joint, in consequence of which these phalanges were not in sufficient contact with the plate.

Temperature.—Between 98° and 99° F. all the time, except for two days, about five weeks after admission. On each of these days it rose to 103°F. (102.4° and 103.8° F.) and then was again normal. (See note made December 30th.)

November 26, 1909: *Complement Fixation Test, Noguchi.*—Negative. Examined by Dr. Kaliski.

The patient made a good recovery, the motion and contour of the joints being little impaired. In three weeks there was a mild recurrence of the chorea and joint swellings, which again soon subsided. It will be at once apparent that this form of articular affection resembles closely a rheumatic attack, but differs in the fact of a general sluggishness of the process, the glandular enlargements, the chorea mild in degree, the cardiac involvement, and the nodules suggest the rheumatic infection. The case takes a mid-ground position between the acute articular rheumatic forms of arthritis and those described in the first part of this paper as a chronic infectious rheumatoid arthritis.

DISCUSSION.

DR. ABT.—I would like to ask the writer if he takes any stock in the neurogenous theory proposed by Still and others?

DR. KOPLIK.—I can scarcely see how the course of the disease supports the neurogenous theory of Charcot.

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AN EXPERIMENTAL STUDY ON THE FOOD REACTIONS IN THE INFANT'S STOMACH COMPARED WITH THOSE IN VITRO.*

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Because of the many discordant views concerning the digestive processes in the infant's alimentary canal which have grown out of purely empirical observations and the comparatively small number of direct investigations upon the infant's stomach, we became interested in finding out for ourselves with the material at hand how the food reactions in the stomach compare with those *in vitro*. In order for an investigation of this sort to be of any practical value it became necessary for us to determine, first, the stimulating effect of food fed to normal infants on the activities of the stomach; second, how these functions are altered by the use of certain chemical substances and how the reactions of these compare with changes obtainable in the test tube with artificial gastric juice.

We have determined (1) that the infant's stomach is capable of stimulation from the first day of life; (2) that unquestionably acid and the ferments are all secreted at this time. Briefly stated, we have obtained rennet curds, an increase in the acidity of breast and modified milk after it has remained in the stomach a certain time and which, by means of Sjöqvist's test is proven to be combined hydrochloric acid, and we have obtained digestion of egg albumen in stomach juice rendered acid to Günzberg's reagent and dimethyl amido azo benzole.

The Secretion of Acid.—As soon as food enters the infant's stomach acid begins to be poured out. If, after a feeding, small

* From the Pediatric Clinic of the University of Michigan Hospital.

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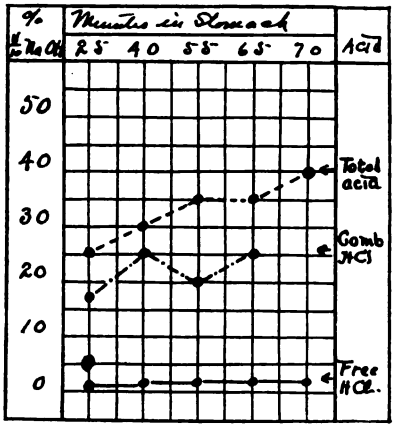

Chart 1. A Normal Acidity Curve.

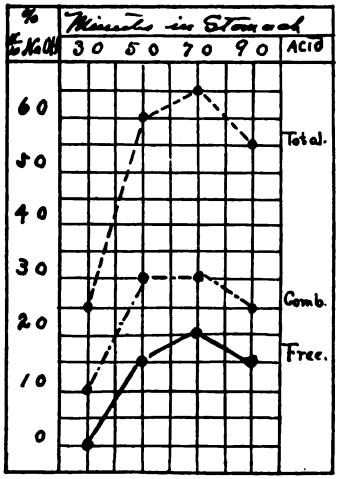

Chart 2. An Abnormal Acidity Curve, Hyperchlorhydria.

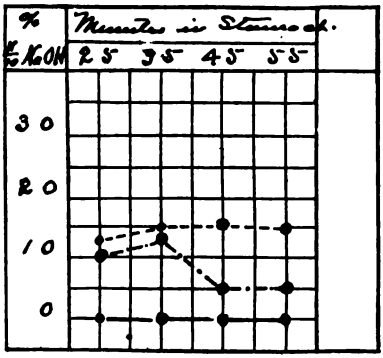

Chart 3. A Normal Acidity Curve.

quantities of stomach fluid are removed from the stomach by means of a soft rubber tube it will be seen that the acidity gradually rises toward a point which may be called the height of digestion and then gradually falls (Charts 1, 2, 3), simulating to a certain extent exactly what takes place in the adult stomach. The secretion of acid in the infant's stomach differs only in degree, for it may be said to be the rule that all the acid combines with the proteid and does not appear in the free state. Only comparatively rarely do we find a case showing free acid, and in this respect our experience is in exact accord with that of Clark and others. Our cases for the greater part varied between the ages of one day and two months; Clark's from two months to eight months. Some of our cases were nine, ten, eleven, twelve and thirteen months old.

Incidence.—In 14 breast-fed infants (Table 1) free acid was detected in 1 (21 analyses). The total acid varied between 10 and 50. In 28 hand-fed infants free acid was detected in 5 (87 analyses) (Table 1). The total acid varied between 3 and 65. In cases showing free acid the amount varied from a trace to 25. In the breast-

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fed case the free acid was +, not enough being recovered to estimate the per cent. The free acid did not necessarily occur in the empty stomach. We have examined many empty stomachs without detecting free acid. We have also detected it several times. In the fasting stomach—the stomach which has received no food for from six to eight hours—we obtained, in 5 infants examined, free acid three times, no free acid twice. We are inclined to believe that infants showing free acid values as high as 10 are in all probability cases of hyperacidity.

The Secretion of the Enzymes.—As stated before, rennet curds can be detected in the breast-fed or hand-fed infant as early as the first day of life.* We have observed that human rennet coagulates both human and cow's milk, while commercial rennet coagulates only cow's milk. Hence the general statement so frequently made that rennet does not coagulate woman's milk. Pepsin is rarely present in the infant's stomach during the first year of life, pepsinogen is almost invariably present. Accordingly, peptic digestion rarely takes place in the infant's stomach, for pepsin does not act in the absence of free hydrochloric acid. It must be remembered that ordinary tests for secondary albumoses and peptones are of no utility in the infant's stomach because such reactions can easily be demonstrated in cow's milk. At the present time only peptone nitrogen estimation by the Kjeldhal method is of any importance. Clark† found this factor practically *nil* in normal infants.

Incidence.—In all infants examined the curdling ferment was present. In all cases excepting those which gave a reaction to free HCl, pepsin was absent. In all cases at some time pepsinogen could be demonstrated by Mett's method.

The Secretion of Mucus.—Mucus is a normal constituent of the infant's gastric contents. It increases in quantity proportionate to the length of time food remains in the stomach. It is always greater toward the end of digestion. The high total acid values sometimes obtained in an infant's stomach contents may be due to the presence of mucus, and are, accordingly, only apparent. If care is taken to select two samples from the same stomach fluid, one free from mucus, the acidity of the sample containing mucus

* In addition to analysis 97, Table 1, we have tested other babies during the first day of life with woman's milk and diluted cow's milk, and have found curds present.

† "The Effect of Certain So-called Milk Modifiers on Gastric Digestion in Infants," *American Journal Medical Science,* 137, p. 827, 1909.

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No.	Case	Age M.	Age D.	Formula F	Formula S.	Formula P.	Formula Sodium Citrate, grs.	Formula Lime Water, %	Formula Whey	*	Motor Tests Time in Stomach	Motor Tests Amount Taken, c.c.	Motor Tests Amount Recovered, c.c.	Lavage	Curds † %	Curds Character	Mucus	Acids Free HCl	Acids Comb. HCl	Acids Total Acid	Date
1	Snyder, Bill	6	8	1	6	1	1		+		2 hrs.	120	75	Few fine curds		Fine, granular, soft	++ thick§	0	16	18	9- 2-09
2	" "		12	2	6	1	1		+		2 "	150	45	" " "		" " "	+ stringy	0	8	10	9- 6-09
3	" "		14	2	6	1	2		+		2 "	150	0	Few floccules			+ thin	—	—	-	9- 8-09
4	" "		21	2	6	1	2		+	Ster.	2 "	165	15			Many small floccules	+ "	0	10	10	9-15-09
5	" "		23	2	6	1		50	+		2 "	120	0	Small amount mucus			+ "	0	+	+	9-17-09
6	Knickerbocker, E.	23		4	5	3.5					2 "	270	0	Few small curds; much mucus			+++ thick	0	—	-	9- 2-09
7	" "			4	5	3.5					2 "	270	0	Large curds; much mucus			+++ "	0	2	3	9- 6-09
8	" "			4	5	3.5					2 "	270	0	Small amount mucus and curds			+ thin	0	—	7	9- 9-09
9	" "			4	5	3.5					1:20	270	0	Few small curds; much mucus			+++ thick	0	—	—	9-13-09
10	" "			4	5	3.5					45 m.	195	0	Few curds; small amount mucus			+ thin	—	—		9-14-09
11	Rebolt, Olive	11	10	3	6	1				R W	2 hrs.	240	0	Small amount mucus; few fine curds			+ "	—	—	-	9- 3-09
12	" "			3	6	1				"	2 "	270	0	Few large curds			+ "	0	+	+	9-21-09
13	" "			3	6	1		40		"	2 "	270	90	Milky material	33	In washings		4	10	14	9-22-09
14	" "			3	6	1				"	1 "	270	52	Clear	23	Not noted	+ thin	0	19	20	9-24-09
15	" "			3	6	1				"	1 "	270	80	Much curds; mucus	30	Large, free from whey	++ thick	0	20	20	9-28-09
16	" "			3	6	1		33⅓		"	1 "	270	58	Few curds		Flocculent curds	++ thin	0	4	4	9-29-09
17	" "			3	6	1		33		"	1 "	240	50	Sour curds		Thick, soft curd	+ thin	0	9	10	9-30-09
18	" "			3	6	1		18		"	1 "	270	125	Few curds		Few small, free from whey	+ "	0	9	9	10- 5-09
19	Holtcamp, Boy	5		2.5	6	1			+	Gav.	2 "	150	40	12 c.c. curds		Medium, soft curds	++ thick	0	10	12	9-29-09
20	" "			2.5	6	1			+	"	2 "	150	34	Few curds		Small curds, mostly whey	++ "	0	20	20	9-30-09
21	" "			2.5	6	1			+	"	2 "	150	45	6 c.c. curds		Few small curds, soft	+ thin	0	18	18	10- 1-09
22	" "			2.5	6	1	Peptzd		+	"	2 "	150	100			Much soft, viscid casein, mostly whey; no distinct curd		0	—	20	10- 4-09
23	" "			2.5	6	1			+	"	2 "	150	50	4 small curds		Few fine curds and whey		0	18	18	10- 5-09

* Abbreviations: Ster.—sterilized. Past.—pasteurized. R·W—rice water. Gav.—food given by gavage. Peptzd—peptonized.
† Percentage of curd recovered estimated by centrifugalization in graduated tube. § See Chart 4.

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24	Williams, Harold	5	4	3	6	1					1 hr.	210	35			Large, blocking tube; whey		0	10	10	10-22-09
25	" "		9	3	6	1					1 "	210	102	Few curds		Medium and small, thick; whey	+ thin	0	10	10	10-27-09
26	" "		10	3	6	1					1 "	210	45	30 c.c. more returned than given; 5% curds		Large curds	+ + thick	0	14	14	10-28-09
27	" "			3	6	1		16⅔			1 "	210	110	Few floccules; curds		Many small curds; whey		0	6	6	10-28-09
28	" "	5	11	3	6	1		16⅔			1 "	210	140	1% fine curds		Many small, soft curds and whey		0	8	8	10-29-09
29	Ebright, Howard	12		3	6	1.5					1:40	240	40			Thick, blocking tube		0	24	24	11- 2-09
30	" "			3	6	1.5					2:15	240	0	5 c.c. curds			+ thin	0	+	+	11- 3-09
31	Keller, Howard	24	8	4	5	3 5					1:45	270	20	Clear		Clear whey; few fine curds		0	26	26	11- 2-09
32	" "		3	4	5	3 5					2:15	270	0	Two small curds				0	+	+	11- 3-09
33	" "		3	4	5	3 5					0:45	270	30			Clear whey; curds block tube		0	26	26	11- 3-09
34	Ebright, Howard	12		3	6	1.5					0:45	150	57			Clear whey; thick, large curds blocking		0	24	24	11- 4-09
35	" "			3	6	1.5					0:30	240	65			Tube blocked with large curd		0	16	18	11- 5-09
36	Case, Charles	14	3	Breast							2 hrs.	?	40	2% curd		Thick and heavy	+ thin	0	10	10	11- 5-09
37	Wolzock, John	4	21	3	6	1.5	2				3 "	150	3	Slightly milky		Fine, small granular curds	+ "	0	—	20	11- 9-09
38	Ebright, Howard	12		3	6	1.5		25			1 "	150	10	10% curds		Thick, curdy material; no whey	+ + thin	0	30	40	11- 9-09
39	" "			3	6	1.5		30			1 "	240	12	½% curds		Thick, large, clear whey	+ + "	0	—	14	11-12-09
40	" "			3	6	1.5					3 "	240	20	3% curds & milky sub.	50	Small and medium sized	+ + thick	0	16	28	11-10-09
41	" "			3	6	1.5		22			1 "	240	42	5% curds & milky sub.		Many small and medium curds; whey	+ thin	0	7	11	11-.0-09
42	Adams, Jack		3	Breast							2 "	?	23	Milky; few fine curds	60	Large, yellow and milky material		0	34	48	12-28-08
43	" "		5	"							2 "	?	28		50	Thick, soft curds and milky material	+ + thick	0	20	35	12-30-09
44	Lane, Baby		8	"							2 "	?	9	Milky	20	Soft white curds; clear whey	+ + "	0	14	20	12-23-09
45	" "		14	"							2 "	?	9	Milky material		Thick curds	+ + + thick	0	—	85	12-29-09
46	" "		15	2	6	.5					3 "	45	0	Few fine curds			+ thin	—	—	—	12-30-09
47	Denney		4	Breast							2 "	?	26	" " "	30	Medium sized, milky whey	+ "	0	15	25	12-23-09
48	Adams, Jack		4	"							½ "	?	Few drops		+			0	—	—	12-29-09
49	" "		4	"							1 "	?	Few drops		+			0	—	—	12-29-09
50	Lane, Baby		14	2	6	.5					1 "	?	—	10% curds				0	—	—	12-29-09
51	Bay, Baby	1		?	?	?					1 "	60	8			Curds not estimated	+ + + thick	0	25	35	11-24-09
52	Denney, Baby		3	Breast							1 "	?			14	Fine		0	—	50	12-22-09
53	Gaus, Baby		17	"							1 "	—	—			Large	+ + thick	0	5	15	12- 1-09

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No.	Case	Age		Formula							Motor Tests				Curds			Acids			Date
		M.	D.	F.	S.	P.	Sodium Citrate, grs	Lime Water, %	Whey	*	Time in Stomach	Amount Taken, c.c.	Amount Recovered, c.c.	Lavage	%	Character	Mucus	Free HCl	Comb. HCl	Total Acid	
54	Wilder, Baby		16	2.5	6	1					1 hr.	75	§ Samp.		20	Medium sized	++ thick	0	—	35	2-28-10
55	Rodda		16	Breast							1 "		"		22	Granular	+ thick	0	—	35	2-28-10
56	Scofield	1	12	3	6	1					1 "	105	"	Clear	14	Medium; clear whey	+ "	0	—	20	2-28-10
57	Grimes		6	Breast							1 "		"		12	Fine, granular	+ "	0	—	55	2 28-10
58	LaPort		17	2.5	6	1					1 "	45	"		15	Thick, blocking tube; clear whey	+ "	10	—	60	2-28-10
59	Youngs	8		2 5	6	1					1 "	45	"		12	Medium and large, blocking	+ "	0	—	40	2-28-10
60	Clark		18	2.5	6	1					1 "	45	"		40	Large and soft	+ "	0	—	45	2-28-10
61	Kirela		12	Breast							1 "	?	"		25	Fine, soft	+ "	0	—	25	2-28-10
62	Young, Leon	7	3	3	6	1					3 hrs.	120	22	Practically clear		Fine, soft; clear whey	+++ thick	15	20	50	2-3-10
63	" "	7	3	3	6	1					1 hr.	90	8 c.c. Samp		30	Fine, soft, floating in whey	++ thick	25	30	65	2-3-10
64	" "	7	4	3	6	1					6 hrs.	120	1		0	No curds	++ "	0	0	0	2-4-10
65	" "	7	4	3	6	1	4 5				1½	90	Samp		8	Granular	+++ very thick	25	20	65	2-4-10
66	LaPort		18	2.5	6	1					1½	45	7 Samp.		25	Small, soft	++ thick	++	—	50	3-1-10
67	"		25	2.5	6	¾		54			—50	90	3 Samp.		30	Medium sized	+++ thick	0	—	15	3-8-10
68	Clark, Raymond		26	2.5	6	1.5			+		—48	90	9 Samp.		15	Very fine; whey milky	+ thin	0	—	18	3-8 10
69	Young, Ronald		26	2 5	6	¾		54			—55	90	10 Samp		40	Medium size	++ thick	0	—	12	3-8-10
70	Wilder, Elmer		25	2.5	6	¾					45	90	11 Samp.		25	Small and medium	++ "	0	—	15	3-8-10
71	Young, Ronald		27	2.5	6	¾	3				1—¼	90	12 9	Little mucus	30	Very fine, granular	+++ "	0	—	15	3-9-10
72	Wilder, Elmer		26	2 5	6	¾	3				1—¼	90	4		40	" " "	++ "	0	—	30	3-9-10
73	Clark, Raymond		27	2 6	6	1 6	3				1—¼	90	4		20	Very fine, milky, well mixed with whey	+++ "	0	—	30	3-10-10
74	Wilder, Elmer		27	Beef broth							1	90	7	Clear			+++ "	0	—	30	3-10-10
75	Young, Ronald		28	" "							1	90	11	"			+++ "	0	—	30	3-10-10
76	Clark, Raymond		28	" "							1	90	13	"			+++ very thick	0	—	26	3-10-10
77	LaPort		28	" "							1	60	2	"			++ thick	0	—	35	3-10-10

§ No effort made to empty stomach.

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78	Young, Ronald		35	2 5	6	¾	1			Past.	1¼	90	15		35	Fine, granular, soft	++ very thick	0	–	18	3-17-10
79	Clark, Ray		35	1	6	1				Na_2Co_3 1gr	Na_2Co_3 1¼	75	11		30	Small; milky material; no whey	+++ " "	0	–	20	3-17-10
80	LaPort		35	2.5	6	¾	1				1¼	75	14		5	Very fine, granular; no whey	++ thin	0	–	12	3-17-10
81	Wilder, Elmer		34	2.5	6	¾	1				1¼	90	11.5		40	Fine, granular; clear whey	+ thick	0	–	30	3-17-10
82	Kolbfleisch		9	?	?	?					½ hr.	?	9	Clear				0	–	30	?
83	LaPort		41	2.5	6	¾					60	75	15	"	20	Medium and small; whey	+ thick	0	–	16	3-24-10
84	Wilder, Elmer		41	3	6	1					63	90	17	"	35	Small, soft; not free from whey	++ "	0	–	9	3-24-10
85	Rodda		40	2 5	6	¾	3				65	75	13	"	40	Very fine, like thick milk; no whey	+ quite thick	0	–	20	3-24-10
86	Grimes		30	1.5	6	½				Past.	60	75	15	"	10	Medium and small; whey clear	++ thick	0	–	16	3-24-10
87	Bently		10	Breast							60	?	16	"	22	Medium and fine; whey milky	+ thin	0	–	18	3-25-10
88	Weston		7	"							62	?	14	"	39	" " small; " "	++ "	0	–	24	3-25-10
89	Russell		10	"							64		14	"	28	Small; whey clear	+ "	0	–	34	3-25-10
90	Reed		55	Skim milk			I.H_2OI				60	60	22	"	2	Medium and small; whey milky	+++ thick	0	–	40	3-31-10
91	Grimes		36	" "							62	75	24	"	20	Large and medium	++ thin	0	–	22	3-31-10
92	Wickham		3	Breast									1	"	–	Only mucus	+++ thick	0	–	–	3-31-10
93	Reed		60	2	6	½			+		70	75	40	"	15	Fine, soft; clear whey	++ thin	0	–	26	4- 4-10
94	Grimes		41	1 5	6	½			+		65	75	11	"	10	Medium and small; clear whey	+++ thick	0	–	36	4- 4-10
95	Peru		13	2	6	½			+		70	60	1	"	–	Small, few	++ "	+	–	–	4- 4-10
96	Wickham		6	Breast							80	–	1	"	–	No curds	+++ "	+	–	–	4- 4-10
97	Jones		1	"							75	–	½	"	–	Traces	+++ "	0	–	–	4- 4-10
98	"		2	"							40	–	3 5	"	60	Deep yellow, medium size	++ "	0	–	15	4- 5-10
99	Wickham		7	"							40		2	"	+	Distinct yellow curd	++ "	0	–	–	4- 5-10
100	Peru		18	2	6	½			+		35	60	11	"	40	Fine, soft curds	++ "	0	–	30	4- 6-10
101	"		18	2	6	½			+		80	60	6.5	"	35	Small, soft curds	+++ "	0	–	55	4- 6-10
102	Reed	2	2	2	6	1			+		1½	75	9	"	20	" " "	++ "	0	–	18	4- 8-10
103	Bently		28	2	6	½			+		1–50	90	2.5	"	1	" " "	+++ "	20	–	55	4-11-10
104	Curtis		4	Breast							1–	–	18	"	50	Medium sized curds	++ "	0	–	20	4-12-10
105	Wickham		14	1.5	6	1.5			+		1–35	60	1	"	+	Two small curds	+ "	0	–	-	4-12-10
106	Bently		29	2	6	1			+		1–40	90	1	"	+	Few small curds	++ "	0	–	-	4-12-10
107	Oldenburg		8	Breast							1–40		2		+	Few large curds	++ "	0	–	–	4-12-10

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will appear higher when titrated against phenolphthalein. The progress of the secretion of mucus is graphically represented in a rather rough manner by the accompanying curves,* (Chart 4), which simply represent our interpretation of the comparative amount found. They refer more to the physical state of the mucus than to the actual quantity. With a well-washed stomach as a basis for our experiment we start with no mucus; in a short time a slight stickiness develops; this becomes stringy, then thick, and, finally, very thick and plug-like. This is true of breast- and hand-

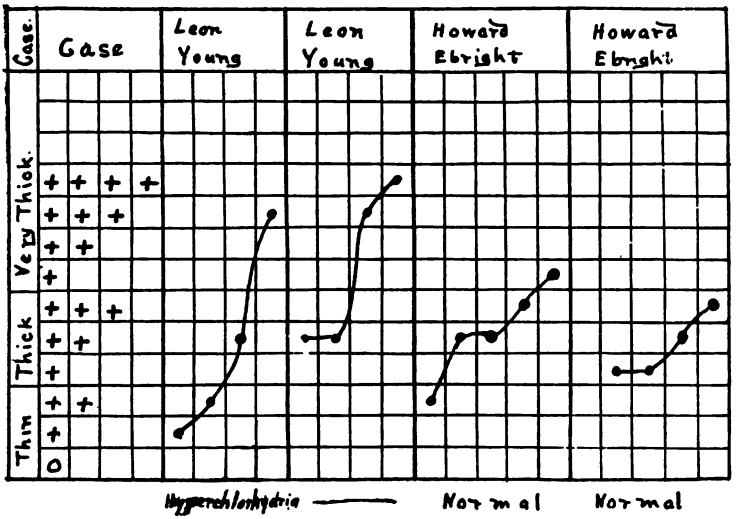

Chart 4.

fed stomachs. When the acidity is greater mucus is often increased.

Basic Calcium Casein Feeding (Lime Water).—When calcium oxide (lime water) is added to calcium casein (milk) in sufficient amount to neutralize any acid present and saturate the proteid until an alkaline reaction to phenolphthalein is obtained, basic calcium casein is formed. So long as this basic condition is maintained neither commercial rennet nor that obtained from human gastric juice can produce coagulation. This is the condition kept up in the test-tube experiment. Reasonable incubation, varying temperature, motion or rest will not bring about coagulation. The amount of calcium oxide necessary to produce this basic condition is not fixed. It varies within wide limits, depending upon the concentration and the acidity of the mix-

* We found no viscosmeter suitable for this work.

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ture. Not infrequently, in ordinary so-called good milk, as high as 60 per cent. of lime water may be necessary to neutralize a 3-6-1 mixture.

If for any cause this basic condition is disturbed as by the addition of dilute hydrochloric acid or the development of lactic acid fermentation, which, however, seldom, if ever, occurs in the normal infant's stomach, rennet action is prompt and certain. It is not necessary for an acid condition to have been reached, only enough is required to neutralize the excess which has been added and which is represented by the difference between an alkaline reaction to litmus and phenolphthalein. It must be remembered that much of the acidity of milk is only apparent, and is due to combination of the standard solution with the proteid (alkali proteid). Neutral calcium casein (amphoteric milk) is coagulated by rennet. These are the conditions obtained in the infant's stomach. As soon as food enters the stomach all its activities are stimulated. If the food be basic calcium casein, sooner or later the excess of alkali is taken up by the hydrochloric acid secreted and the calcium casein is in turn acted upon by rennet. There is, accordingly, a period during the sojourn of basic calcium casein in the infant's stomach when it remains intact, and during this time, whatever its action may be, it is exerted

TABLE 2

Control.									Basic Calcium Casein							
Case	Control Formula	Amount taken, c. c.	Amount c. c. recovered	Time in stomach.	Character of Curds	Acids Free	Acids Combined	Acids Total	Per cent. of lime water	Amount taken, c. c.	Amount recovered	Time in stomach	Character of Curds	Acids Free	Acids Combined	Acids Total
Snyder	2 6-1	120	15	2 h.	Few fine curds	0	16	18	50	120	0	2 h.	Little mucus in washings	0	0	0
Rebolt	3-6-1-RW	240	0	2 "	Three small	0	0	0								
		270	15	2 "	Few small & large	0	+	+	40	270	90	2 "	Thick milky material	4	10	10
		270	52	1 "	Small with whey	0	19	20	23	270	58	1 "	Flocculent curds	0	4	4
		270	95	1 "	Granular & whey	0	20	20	23	270	60	1 "	Thick soft curds	0	9	10
									18	270	125	1 "	Few small curds & whey	0	9	9
Williams	3-6-1	210	37	1 "	Many large curds	0	18	18								
		210	102	1 "	Many large and medium	0	10	10								
		210	49	1 "	Large blocking	0	14	14	12½	210	141	1 "	Many small feathery curds	0	8	8
Ebright	3-6-1½	240	40	1¾	Large blocking tube	0	24	24	22	240	97½	1 "	Many small and medium curds	0	7	11
		150	57	¾	Large blocking	0	16	18	30	240	13	1 "	Large thick sour curds	0	30	40
		240	65	⅛	Large and small	0	24	24	25	90	22	1 "	Thick milky material	0	—	14

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upon the stomach functions. This period varies, depending upon the secretion of acid. It is shorter in hyperchlorhydria, longer in hypo and achlorhydria. In one case we found no curd formation until forty minutes, while at forty-five minutes a large amount of large curds was obtained. The stomach seemed full of clear, thin, milky fluid at forty minutes. At forty-five minutes only 20 c.c. of this curd material was recovered (acid opening).

Conditions produced in the test tube with rennet solution and basic calcium casein do not obtain in the stomach, for in all instances curds are formed. (Table 2.) These curds vary in their consistency from a thick, milky material, which may be separated by rapid centrifugalization, to large and thick curds, which in our experience were always soft. One has to ask the question, Are these rennet curds (calcium paracasein), or are they acid curds (casein hydrochlorid). When basic calcium casein remains in the stomach sooner or later a point is reached when the gradual pouring out of acid has neutralized the excess of calcium but has not yet united with all the soluble calcium salts. During this stage, rennet, which is ever present, may unite with the now neutral calcium casein to form calcium paracasein curd. If, as is generally the case, acid continues to be poured out, it unites with the calcium paracasein curd to form paracasein hydrochlorid and calcium chlorid. These successive changes must have taken place before hydrochloric acid appears in the free state. The curd found after lime water must be either calcium paracasein or paracasein hydrochlorid, and these are differentiated from each other by their physical characteristics and their solubility in warm 5 per cent. salt solution.

Calcium paracasein curds are tough ductile curds soluble in warm 5 per cent. salt solution.

Paracasein hydrochlorid curds are granular soft curds insoluble in warm 5 per cent. salt solution, but which after washing in water are soluble in weak ammonia water, from which they may be reprecipitated by addition of weak acid.

In our experience the latter curd is the one found in the stomach after feeding basic calcium casein.

The Effect of Basic Calcium Casein on Gastric Acidity.—The acidity of the stomach after a basic calcium casein meal very seldom reaches the acidity obtained after a control meal. The acidity is almost invariably distinctly lowered. (Table 2.) The average total acidity in the control series is 18.4, in the basic series

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13.6. Free acid was detected once in the basic series, and in one test the total acid went as high as 40. If this one meal is omitted, the average acidity of the basic series drops to 9.8 as compared with 18.4 in the control. Free acid seldom occurs after a basic calcium casein meal, but regardless of the per cent. of calcium oxide added the proteid becomes saturated with acid. In this respect our results appear to differ from statements made that lime water stimulates excessive flow of acid. This may be true if we take into consideration the amount of acid that is necessary to neutralize the excess of acid-binding substance present in a basic meal. But this does not concern us clinically—it is the proteid combined acid and the free acid which exert their effect upon the subsequent food reactions in the stomach.

The Effect of Basic Calcium Casein on Stomach Motility.—Basic calcium casein does not tend to promote stomach evacuation, as is generally believed. Our cases (Table 2) are too few to warrant us in making a general statement. They are of sufficient number, however, to give the doubt to the generally accepted theory, and they bear out what one must expect if modern physiology, advanced by experimentation on the lower animals, holds true.

The stomach of a healthy infant must be as near a normal organ as can be found during life. It is, therefore, especially well adapted to physiologic research. Psychic elements do not enter into the case, the infant has no fears and it is not hard to find one who will sleep through the entire examination, which may last over an hour. Accordingly it is reasonable for us to expect, if the physiology of man and animals is similar, the pyloric mechanism to respond to the same stimuli as, for example, that of the cat. Particularly through the experiments of Pawlow and Cannon we are led to believe that the pylorus is controlled by acid. Acid opens it, and when acid reaches the duodenal mucous membrane the nervous mechanism which has to do with the closure of the pylorus is brought into action, and is sooner or later overcome by the dissipation of the duodenal acidity. Hence theoretically we must expect slowing in a stomach evacuation after alkaline food, because acid stimulation of the pylorus is thus delayed.

It has been a difficult matter for clinicians to harmonize their clinical findings with the newer physiology, for in the secretory neuroses of the adult, as well as the infant, exactly opposite con-

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ditions seem to exist. In achylia we have rapid evacuation; in hyperchlorhydria the tendency very often, indeed, is to delayed evacuation. We have conducted two experiments with the hope of getting some light on this question, in both of which the infants were admirably well adapted for such observations because of their good health and their indifference to the passage and retaining of the tube. Unfortunately one left the hospital before our work was complete.

We selected as a test meal one free from fat, thus eliminating a factor which is known to delay stomach evacuation in animals, and which has been shown by Cowie and Munson* to produce a similar effect in man. Skim milk diluted one-half with water was employed. After several trials we selected sixty minutes as the best period to allow the food to remain in the stomach in Case 1, forty-five minutes in Case 2. With control meals, the acidity of which varied between 6 and 8, the largest amount recovered at the end of the period was 3 c.c. to 10 c.c. We took great pains to have the amount fed, 75 c.c., and the time of sojourn in the stomach, forty-five minutes, constant factors. To accomplish the latter it was necessary to introduce the food by gavage, which was in each case accomplished in the same number of minutes. The acid meal was prepared by titrating with dilute hydrochloric acid (10 per cent.) solution until the acidity desired was obtained. The food was then kept at body temperature for one hour to assure complete combination of acid with proteid. And again in the case of the free acid meal, retested and treated if necessary, to assure the acid value as it entered the stomach. Our cases were so well controlled that although the records of the amounts recovered after the allotted time cannot be said to be absolutely accurate, we think the error varied not more than 1 or 2 c.c. After syphoning off the stomach contents the stomach was washed with a definite amount of warm water, which in all cases was recovered. Any additional amount recovered was calculated as original stomach contents. In an investigation of this character gross differences alone can be considered. The position of the infant was constant in all experiments. (Tables 3 and 4, Charts 5 and 6.)

Taking Case 2 we find that in the control meal only 2 or 3 c.c. remained after forty-five minutes. When free acid is given from 28 to 45 c.c. are recovered. When the total acidity of the meal

* "An Experimental Study of the Action of Oil on Gastric Acidity and Motility," *Arch. Int. Med.*, Vol. I., p. 61, 1908.

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TABLE 3. CASE 1. ACID CONTROL OF PYLORUS.

Meal	Acidity of Meal	Amount Taken	Amount Recovered	Characters	Free HCl.	Total Acid
Control		75 c.c.	6.5 c.c.	Fine curds and clear whey; mucus, +++ thick	0	10
Control		75 c.c.	10. c.c.	20% medium and small curds, clear whey; mucus +++ thick	0	14
Acid	10 - —	75 c.c.	37. c.c.	10% very fine granular curds, whey milky	0	54
Acid	4 - 80	75 c.c.	38. c.c.	18% fine granular curds, whey milky; mucus, +++ thick	4	68

TABLE 4. CASE 2. ACID CONTROL OF PYLORUS.

Meal	Acidity of Meal	Amount Taken	Amount Recovered	Characters	Free HCl.	Total Acid	Date
	Free Total						
Control	0 - 6	75 c.c.	3 c.c.	Small soft white curds; mucus, +++ thick	0	15	4-11-10.
Control	0 - 8	75 "	0 "	Wash water contains only mucus, +++ thick	-	—	4-13-10.
Acid	10 - 60	75 "	45 "	Large and medium curds soft; mucus, +++ thick	2	40	4-12-10.
Acid	4 - 80 - 76	75 "	38 "	18% fine granular curds; mucus, +++ thick	4	68	4-
Acid	10	75 "	27 "	14% fine granular curds; mucus thin	8	68	4-14-10.
Acid	0 - 75	75 "	25 "	40% large granular curds; same as before taken	2 0	36	4-15-10
Acid	0 - 30	75 "	4 "	2½% fine green curds; mucus +++ thick	0	35	4-18-10.
Acid	0 - 30	75 "	0 "	Washing clear, few flecks of curd	-	—	4-20-10

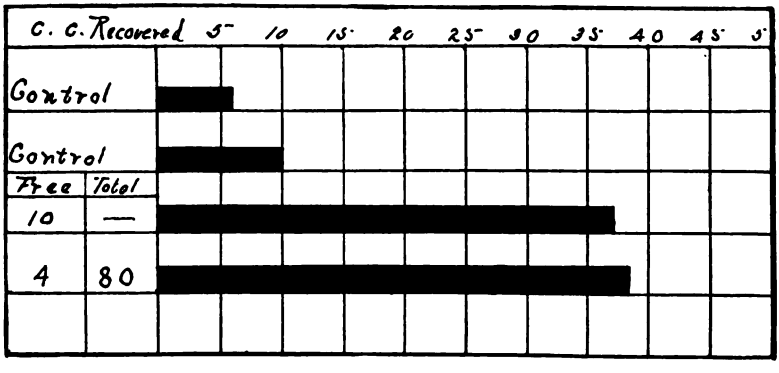

Chart 5, Case 1. Acid control of pylorus.

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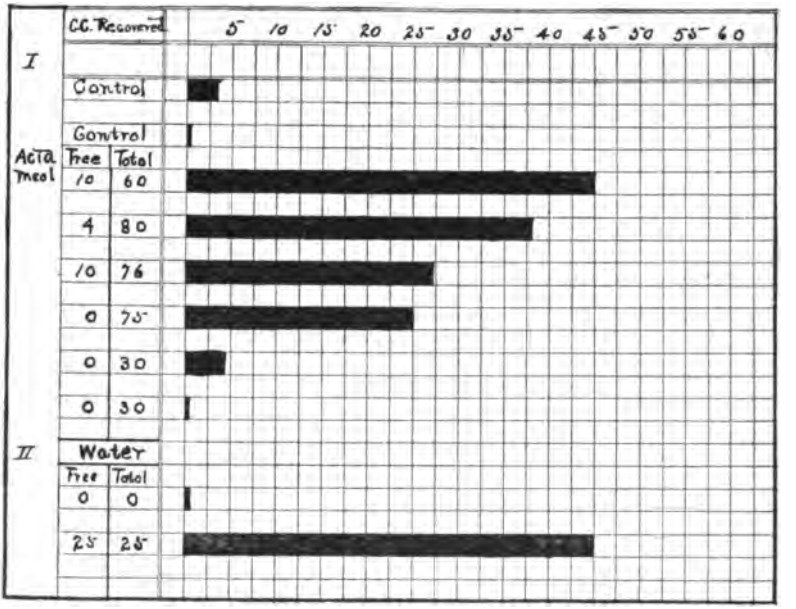

Chart 6, Case 2. Acid control of pylorus.

without free acid is 75, 25 c.c. are recovered. When the acidity of the meal is 30 the evacuation of the stomach is the same as in the control. In the instances of delayed evacuation we have conditions comparable to hyperchlorhydria in the adult. In those instances where the acidity is low we have conditions in a way comparable to achlorhydria in the adult. In other words, the manner of evacuation of the stomach (with this meal) depends upon the degree of acidity present. We also found that by rendering water acid its sojourn in the stomach could be lengthened. (Chart 6, II.) The delay of evacuation must be due to one of two factors:—

(1) Either the discharge of a large amount of acid into the duodenum requires a longer time for its neutralization, hence the duodenal closing reflex is prolonged, or (2) the large amount of acid on the stomach side produces spasm of the pyloric sphincter.

The question—Is the delay of evacuation in the infant's stomach after an acid meal due to the presence of high acidity to the left or right of the pylorus?—remains to be settled.

The Effect of Basic Calcium Casein on Stomach Motility.—So far as our experience takes us we are led to the opinion that basic calcium casein does not tend to promote rapid evacuation

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of the stomach in all cases as is generally believed. Our cases are too few to warrant us in making a general statement. We report ten control meals and nine basic calcium casein meals in three babies. The evacuation was the same or delayed five times and increased twice by basic calcium casein as compared with the control meal. If we mass these we find the average amount recovered in the controls 52.7 c.c., in the basic meals 67.4 c.c.

Citrated Milk.—When oxalates and fluorides are added to milk the milk is said to be decalcified. Combinations thus formed are poisonous and cannot be employed in infant feeding. In 1893 Sir A. E. Wright, of London, recommended sodium citrate as a harmless decalcifier of milk for the purpose of preventing the action of rennet on milk proteid. He advised it in ½ per cent. strength, or 2½ grains to the ounce (*Lancet,* July, 1893). As an illustration of the harmlessness of sodium citrate to young infants, I may relate a mistake which occurred in my wards during the past winter. A new nurse assigned to service in the milk laboratory became confused in the weights; instead of weighing out 9 grains of sodium citrate for the infant's formula, weighed 9 grams. This amount was given for two days before it was discovered by the physician in charge of the case. Aside from a few extra stools the infant manifested no symptoms. Shaw and Wheeler are of the opinion that we do not yet know what chemical change occurs when sodium citrate is added to milk. Sodium citrate unquestionably prevents all rennet action, not only in the test tube, but also in the infant's stomach. The curds formed in the stomach are casein hydrochlorid curds and have the same appearance, except for their admixture with mucus, as those formed in the test tube by careful addition of dilute hydrochloric acid. After carefully washing out the acid with distilled water these curds, whether from the stomach or the test tube, are readily soluble over night in dilute ammonia water. They may be reprecipitated from their alkalin solution, which has an opalescent color, by means of dilute hydrochloric acid. When mucus is abundant complete solution is not so quickly obtained. Usually, however, if the proportions are right, most of the curd has dissolved over night.

Our observations on citrated milk were made chiefly for the purpose of determining the nature and the physical state of the curd; this we found was uniformly casein hydrochlorid, fine, granular and soft as compared with the large curds so frequently

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found after whole milk and sometimes after ordinary modified milk. Our observations on the effect of sodium citrate upon stomach motility and acidity are only incidental. In the few comparisons that can be made from similar citrated and noncitrated meals recorded in the list of analyses (Table 1), one might say that no particular effect upon the acidity occurs. In some cases the acidity is higher in the citrated meals; in others, lower. We have gained the impression that when citrated meals are employed a larger amount of mucus is poured out. Clark investigated the point of acidity and found it lowered after sodium citrate. In the two following experiments, where small samples of stomach juice were removed from time to time, the following

TABLE 5.—CITRATE.

Feb. 3, 1910. CONTROL MEAL.

Leon Young, 7 months.

Time	c.c. Recovered	Time in Stomach		Curds		Mucus	Acids		
		Hrs.	Mins.	%	Character		Free	Comb.	Total
9:20					[Fed 90 c.c. 3.6.1. Total acid 6]				
9:40	7.5		30	35	Medium sized, soft	+ thin	0	15	25
10:00	7 5		50	40	" " "	+ + thick	20	30	60
10:20	8.0	1	70	30	Medium, free from whey	+ + quite "	25	30	65
10:40	6.0	1	30	20	Medium, floating in whey. Stomach empty	+ + + very "	20	25	55

Feb. 4, 1910. SODIUM CITRATE MEAL.

Time	c.c. Recovered	Time in Stomach		Curds		Mucus	Acids		
		Hrs.	Mins.	%	Character		Free	Comb.	Total
8:40					[Fed 90 c.c. 3.6.1. sodium citrate, grs. lvss.]				
9:00	8.0		35	12	Fine, granular, soft, feathery	+ + quite thick	0	15	25
9:20	7.5		55	14	" " " "	+ + " "	0	20	40
9:40	9 0	1	15	30	" " distinct from whey	+ + + very "	15	35	75
10:00	7.0	1	35	8	" " viscid mucus	+ + + + " "	25	20	65
10:20					Stomach empty. Much mucus in lavage; only a few small curds	14%			

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TABLE 6.—CITRATE.

Feb. 16, 1910. CONTROL MEAL.

Howard Ebright, age 16 months.

Time, A.M.	Formula	Amount Fed c.c.	Amount Recovered c.c.	Time in Stomach	Curds %	Curds Character	Mucus	Acids Free	Acids Comb.	Acids Total
9:00	Began to feed									
9:30	Whole milk	240	15	15	3	Size of a pea; free from whey	+ stringy	0	12	28
9:50			24	35	20	Size of a filbert; blocking tube	+ "	0	20	34
10:00			8	55	6	Fine, floating in whey	++ thick	0	25	48
10:30			1	1.15	0	None in wash water. Stomach empty	+++ "	0	—	—

Feb. 17, 1910. CITRATE MEAL.

Howard Ebright.

Time	Formula	Amount Fed c.c.	Amount Recovered	Time in Stomach	Curds %	Curds Character	Mucus	Acids Free	Acids Comb.	Acids Total
8:40	Began to feed	240								
9:00	Whole milk + sodium citrate grs. viii.		13	15	15	Fine granular; not free from whey	+ stringy	0	15	30
9:20			22	35	16	Granular; free from whey	++ thick	0	14	32
9:40			10	55	5	Granular; whey clear	++ "	0	21	36
10:00			8	1.15	?	Few small granules floating	++ "	0	30	55
10:20			0.5	1.35	0	No curds. Stomach empty	+++ thicker	+	—	—

There was slight regurgitation at 9:20 and 9:40. The tube was never blocked. The curds were distinctly soft as compared with the control.

curves were obtained. (Charts 7 and 8.) In the second case we had a patient who manufactured large amounts of acid. This enabled us to get a free acid curve. (Chart 8.) The appearance of free acid is delayed in the citrate meal, but ultimately reaches the same height as the control. The total acidity rises more slowly, but ultimately exceeds the control. In the first case (Chart 7) very similar conditions are present, except that in the citrate meal free acid ultimately appears just as the last food is leav-

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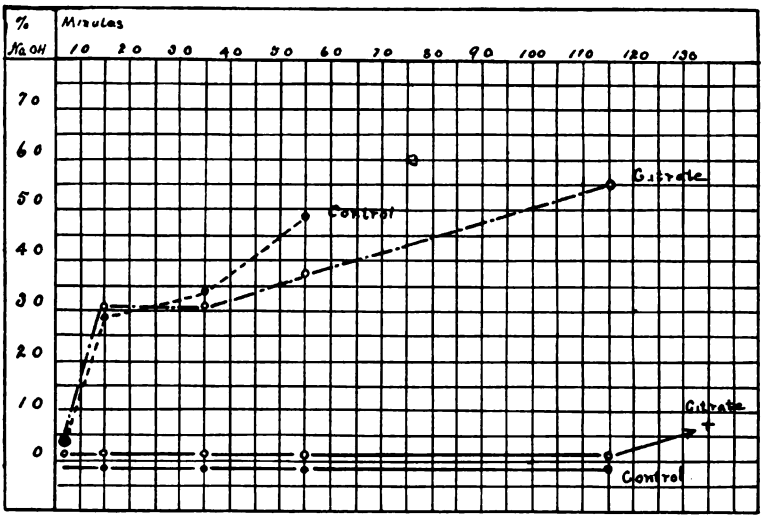
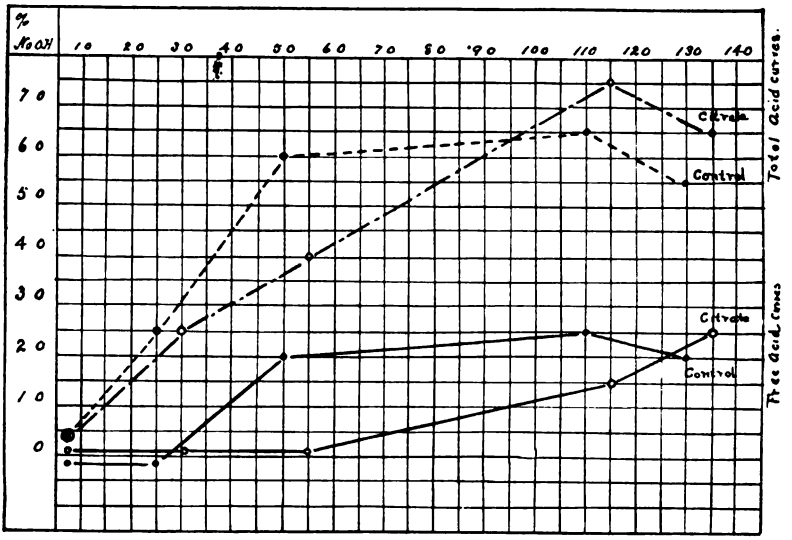


Case of Table 6, showing graphically amount of total acid with citrate and control meals.



Case of Table 5, showing graphically curves for total and free acid for citrate and control meals.

ing the stomach. In the first experiment the amounts withdrawn are not so uniform as in the second. It may be that some knowledge can be gained from experiments of this kind if they are well controlled.

To return again to stomach motility, or the time required for

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a certain amount of food to pass the pylorus, which must doubtless be one of the most important factors in the proper feeding of an infant. It usually concerns us little in the ordinary case, in the case which seems to thrive in spite of bad feeding. In the emaciated case, the case of malnutrition, or the so-called difficult feeding case, we are not really doing our full duty if we do not put ourselves in possession of all the facts in the case. The baby who needs it, and *only* he, is entitled to as careful stomach analysis as is the adult.

If at the beginning of a feeding period we find in the stomach retained food in any appreciable amount, our period between feedings must be lengthened or something must be done to hasten the evacuation of what may seem to be a sluggish organ. A condition of this kind must always be regarded with much care. We must be sure the faulty evacuation is due to so-called sluggishness or atonia and not, in spite of vigorous muscular effort, to a pyloric obstruction. Why should we not have varying degrees of stenosis in an infant as well as in an adult, stenosis of the first and second degree, possibly due to the same cause but producing a different group of symptoms. Enquiry into the motility of our cases may be a potent factor in the recognition of stenosis of the first degree and in the skillful after care of the case.

The evacuation of any stomach, infant or adult, depends upon several factors: (1) The amount of food to be propelled through the pylorus in a given time, (2) the character of the food with regard to the mechanical resistance it offers at the pylorus, (3) the propelling force and the factors which go to make up this force, muscle and nerve, and (4) the integrity of the nervous mechanism of the pylorus.

The first postulation requires no comment. The second is open to discussion. It is evident that a marble will have greater difficulty than a pea in passing an elastic orifice, the size of which without undue stretching is slightly less than the marble. Conditions comparable to this may be considered rare ones in the normal infant's stomach. In the ordinary case the comparative size of the curd, we are inclined to believe, in reality does not have so much to do with the rapidity of stomach evacuation as one might think. Most of the curds in the stomach are soft and capable of being easily crushed and propelled through a patent pylorus by normal muscular effort. If the integrity of the nervous mechanism is intact the chemical condition of the stomach con-

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tents, we think, may be a more potent factor in delayed evacuation than the size of the curd.

CONCLUSIONS.

(1) The infant's stomach secretes all the digestive juices from the first day of life.

(2) Free hydrochloric acid rarely occurs in the infant's stomach during the active part of the day. It may be found at times in the fasting stomach, at the height of digestion, or at the end of the digestive period.

(3) The evacuation of the normal infant's stomach depends, to a large extent, upon the degree of acidity of the stomach contents. Excessive acidity delays evacuation. A total acidity value varying from 8 to 30 offers the normal stimulus for pyloric relaxation.

(4) Basic calcium casein does not inhibit the formation of rennet curds in the infant's stomach. It delays the formation of curd, delays the saturation of the proteid with acid, delays the acid pyloric reflex, and by so doing delays the evacuation of the stomach contents. The action of rennet on basic calcium casein in the test tube is not comparable with that which takes place in the stomach as the conditions are not the same.

(5) Sodium citrate inhibits the action of rennet in the stomach as well as in the test tube. The curd formed in the stomach, and that formed when dilute acid is added to citrated rennet milk, are casein hydrochlorid curds.

(6) The curds normally present in the infant's stomach are paracasein hydrochlorid.

We wish to acknowledge the courtesy of Professor Peterson and members of the maternity staff in permitting us to use some of the infants in the maternity wards.

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THE ESTIMATION OF CHLORIDS IN THE STOMACH CONTENTS FROM NORMAL AND FROM ATROPHIC INFANTS.*

BY A. H. WENTWORTH, M.D.,
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The observations recorded in this paper are a continuation of work briefly alluded to in a previous article.† The purpose of this work is to discover whether or not the hydrochloric acid of the gastric juice is diminished in cases of infantile atrophy.

In the article on the causation of atrophy referred to above, I suggested a theory of the cause of infantile atrophy based on the results of secretin determinations in atrophic and well-nourished infants.

The secretin production from the cases of atrophy which I examined was much diminished compared with that from a limited number of well-nourished infants. The explanation for this diminution in secretin in atrophic infants was sought for higher up in the digestive tract. It seemed improbable that the condition was a primary one in the small intestines. The normal stimulus, according to Bayliss and Starling, which causes the transformation of prosecretin into secretin is supplied by the hydrochloric acid of the gastric juice. It was possible theoretically that the stomachs of atrophic infants secreted a diminished amount of hydrochloric acid and in consequence failed to produce an adequate stimulation of the duodenum. Most of the secretin, according to Bayliss and Starling, is produced in the upper portion of the small intestines. Only a constant and marked diminution in the amount of hydrochloric acid secreted by the stomach would have any value in support of the theory. In any case, the determination of hydrochloric acid was but a link in the chain. The cause for a constantly diminished production of hydrochloric acid by the stomach would have to be sought.

In the first series of observations, the stomach contents were obtained one hour after eating from a limited number of atrophic infants fed on mixtures of cows' milk. The determinations were made by Sjöquist's method.‡ The results showed a diminished amount of hydrochloric acid in the stomach contents in every

* From the Laboratory of Biological Chemistry of the Harvard Medical School. The expenses of this research were defrayed in part by a grant from the Proctor Fund for the Study of Chronic Diseases.

† Wentworth, A. H.: "The Cause of Infantile Atrophy Deduced from a Study of Secretin in Normal and Atrophic Infants," *Journal of American Medical Association*, 1907, Vol. XLIX., p. 204.

‡ Sjöquist: *Ztschr. f. Klin. Med.*, 1897, Vol. XXII.

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case. The same series of observations included determinations of hydrochloric acid in the stomach contents from a limited number of well-nourished and apparently healthy infants fed on mixtures of cows' milk. The results in these cases were variable. In four cases the amount of hydrochloric acid was much greater than in the cases of atrophy. In three cases the amount of hydrochloric acid approximated that of the atrophic infants. As these results were not conclusive, a second series of observations was made with a view to controlling the first series. The same method of analysis was employed.

This article gives the results from the second series of observations and some conclusions that may be drawn from them.

METHOD OF DETERMINATION.

The modified Sjöquist method for the determination of hydrochloric acid in the stomach contents was employed in these analyses. It has been stated that this method is inaccurate when the diet consists of milk, because an unknown quantity of the phosphates which are present in the milk combines with some of the hydrochloric acid to form an insoluble compound that remains in the filter-paper when the barium chlorid is extracted with hot water. This has no particular importance so far as my work is concerned, because I wished to determine whether there was a constant and marked diminution of hydrochloric acid in the stomach contents from atrophic infants compared with that from healthy infants. Such differences to be of value would have to be large enough to place them well beyond the range of error in the method.

Unfiltered stomach contents were used in every case. The stomach contents were preserved in a frozen condition until the analyses could be made. Soft rubber catheters, No. 19 to No. 25 (French), were used to obtain the contents. The solid tips were cut off and in addition a large oval opening was made in one side about one-half an inch from the tip. The edges of these openings were rounded off by heat. The contents were obtained with the infant lying on its side. The quantity of contents obtained does not necessarily correspond to the quantity present in the stomach. In some cases the lumen of the tube becomes obstructed by curds; in other cases the presence of much mucus prevents the contents from being expressed. In a few

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instances there was every reason to believe that the stomach contained a much larger quantity of fluid than was obtained, and yet there was no evidence of obstruction of the catheter by curds or by mucus.

I have arranged in the form of tables what I believe to be the essential data derived from these observations. This method of arrangement facilitates comparison.

The diagnosis of infantile atrophy was made from clinical evidence alone. Most of the cases were under observation for several weeks.

TABLE I.—QUANTITATIVE ESTIMATION OF HYDROCHLORIC ACID IN THE GASTRIC CONTENTS OF INFANTS.*

ATROPHIC INFANTS.

Name.	Age in Months.	Weight.			Food. Per Cent. of.			Quantity.		Free HCl†	Gm. of HCl in 100 c.c. of Gastric Contents.
		Gm.	Lbs.	Oz.	Fat.	Sugar.	Proteid.	Oz.	No. Feedings.		
A.....	4.5	3690	8	2	3	6	1	4	8	0	.01
B.....	3.5	2300	5	0	3	6	1	2.5	10	0	.016
C.....	3.5	3410	7	8	3	6	1	2.5	10	0	.012
D.....	9	3990	8	12	3	6	1	4.5	8	0	.02
E.....	3.5	4030	8	14	3	6	1.25	4	8	0	.041
F.....	3.5	4150	9	2	3	6	1	...	..	0	.07
G.....	15	7550	16	10	‡	..	....	8.5	5	0	.018
H.....	15	6830	15	..	§	..	....	8	6	0	.01+

E. and F., although much under weight, were not typical cases f atrophy. Neither their previous history nor their appearance corresponded to atrophy.

WELL-NOURISHED INFANTS.

K.....	7.75	6360	14	..	—Milk—		....	6	6	0	.16
L.....	8	6780	14	15	4	7	1.25	8	6	¶	.145
M.....	6	5900	13	..	3.5	7	1.8	6	6	¶	.144
N.....	8	6950	15	5	4	7	1.5	6	6	0	.165
O.....	7	5540	11	3	4	7	1.25	5	8	0	.045
P.....	5	5180	11	6.5	3.5	6	1.8	6	6	0	.015
R.....	8	7350	16	3	4	7	1.25	6	7	0	.01+

* From article in *Journal of American Medical Association*, 1907, Vol. XLIX., p. 204.

† The reaction in the atrophic infants was faintly acid, in the well-nourished infants, acid.

‡ Milk, 7 oz.; oat jelly, 1.5 oz.

§ Milk, 4 oz.; barley jelly, 4 oz.

¶ Faint reaction.

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TABLE 2.—QUANTITATIVE ESTIMATION OF HYDROCHLORIC ACID IN THE GASTRIC CONTENTS OF INFANTS

ATROPHIC INFANTS

No.	Name.	Date.	Age.	Weight Gm.	Food. Approximate Per Cent. of Fat.	Sugar.	Protein.	Feedings. in 24 Hours.	Quantity c.c. Each Time.	Stomach Contents. Obtained After Feeding, Hour.	Quantity Obtained, c.c.	Chlorin in 100 c.c.	Remarks.
1	J. McH.....	1/27/08	2 mos.	3010	12 % cream, 150 c.c. Whey..... 600 c.c.			10	75	1	25	.004	.....
2	A. B.....	12/14/07	6+ mos.	3780	3.	6.	1.	8	105	2¼	22	.03	No gain in weight for 7½ weeks.
	A. B.....	12/16/07	6+ mos.	3760	3.	6.	1.	8	105	1½	11	.021	.....
	A. B.....	12/17/07	6+ mos.	3710	3.	6.	1.	8	105	2½	25	.028	.....
	A. B.....	12/20/07	6½ mos.	3730	3.	6.	1.	8	105	1½	57	.006	.....
	A. B.....	1/ 5/08	7 mos.	4040	Breast-milk			8	900 to 1000	1	70	.009	Gained 320 gm. in 1 week.
	A. B.....	1/12/08	7 mos.	4325	Breast-milk			8		1½	45	.016	Gained 285 gm. in 1 week.
	A. B.....	1/19/08	7 mos.	4505	Breast-milk			8	Daily	2	50	.006	Gained 180 gm. in 1 week.
3	C. T.....	1/ 2/08	3½ mos.	2990	3.	6.	1.	8	105	1¼	12	.04	.....
	C. T.....	1/13/08	4 mos.	3010	3.	6.	1.	8	105	1½	52	.01	.....
	C. T.....	1/20/08	4½ mos.	3170	3.	6.	1.	8	105	1¾	12	.03	.....
4	D. K.....	1/25/08	4½ mos.	4270	3.5	6.5	1.	8	150	1	55	.002	Fat intolerance; fat crystals in feces; symptoms subsided on whey mixture.
	D. K.....	1/27/08	4½ mos.	4250	12 % cream, 240 c.c. Whey..... 720 c.c.			8	120	1½	6	.024	.....
	D. K.....	1/29/08	4½ mos.	4230	"	"		8	120	2	29	.002	.....
	D. K.....	1/31/08	4½ mos.	4250	"	"		8	120	1	75	.002	.....
5	D. S.....	1/25/08	3 mos.	3940	12 % cream, 210 c.c. Whey..... 630 c.c.			8	105	1¾	43	.003	Gained 410 gm. in 14 days.
6	C. T.....	1/27/08	5 mos.	3150	12 % cream, 180 c.c. Whey..... 780 c.c.			8	105	1½	15	.046	.....
	C. T.....	1/31/08	5 mos.	3310	"	"		8	120	1	42	.02	.....
7	M. F.....	1/25/08	3½ mos.	3350	3.5	6.5	1.	8	105	1¼	45	.004	Died Feb. 17 of acute miliary tuberculosis; chr. glandular tub.
	M. F.....	1/29/08	3½ mos.	3250	3.5	6.5	1.	8	105	2¼	30	.006	.....

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TABLE 2.—(Continued)

No.	Name.	Date.	Age.	Weight Gm.	Food. Approximate Per Cent. of Fat.	Sugar.	Protein.	Feedings. in 24 Hours.	Quantity c.c. Each Time.	Stomach Contents. Obtained After Feeding, Hour.	Quantity Obtained, c.c.	Chlorin in 100 c.c.	Remarks.
8	R.....	2/26/08	8 mos.	4275	3.5	7.	1.25	4	180	1	90	.005	Stomach contents obtained after modified milk feeding; gained 615 gm. in last 11 days.
					Breast-milk			4					
					WELL-NOURISHED INFANTS								
9	J. H.....	1/23/08	18 days	1900	Whey			10	45	1	5	.01	Premature infant. Breast-milk first 15 days.
10	A. C.....	12/20/07	20 days	2420	Whey			10	60	2	10	.048	Remained in hospital 26 days and gained 430 gm. in same food.
	A. C.....	12/28/07	28 days	....	3.	6.	.75	10	60	1¼	17	.026	.....
11	E. G.....	1/ 7/08	8+ mos.	8000	Breast-milk			5					Infant had pyelitis and lost weight while in hospital. Stomach contents obtained after breast-milk feedings.
					Cow's milk, 120 c.c.			1	180	1½	55	.025	
					Barley water, 60 c.c.								
	E. G.....	1/20/08	.....	....	" "			"	180	1	80	.046	
12	A.....	2/25/08	7 mos.	8550	3.50	6.50	1.50	4	210	1½	75	.04	Wet-nurses' baby (normal).
					Breast-milk			4					
	A.....	2/26/08	7 mos.	....	.....			..	....	1	30	.12	.....
13	M.....	2/26/08	5½ mos.	7290	Cow's milk mixture			4					
					Breast milk			4	....	1	95	.05	.....
14	F.....	2/26/08	6 mos.	7320	Specimen obtained after breast feeding.					1	32	.09	Last 3 wks. supposed to have mod. cow's milk 8 feedings.
15	D.....	2/26/08	3 mos.	5370	Cow's milk mixture			4					Formerly 4 breast and 4 cow's milk. Said to have vomited after last feeding.
					Breast-milk			4	....	1	12	.03	
16	K.....	2/25/08	3 mos.	7260	Breast-milk			..	....	¾	5	.035	.....
17	G.....	2/25/08	5½ mos	6000	Breast-milk (?)			..	....	1	60	.005	Baby fed before coming to hospital; said to be breast-fed.

* Free hydrochloric acid was tested for by Günzberg's test in every case. A *positive* reaction was obtained but once (Case 12).

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DISCUSSION OF DATA.

A possible relation of the amount of stomach contents to the degree of acidity could not be determined accurately, but appeared not to be present in this series. In most instances, repeated examinations of the stomach contents obtained from the same atrophic infant from one to two and a half hours after eating showed little, if any, increase in the hydrochloric acid as the result of prolonged digestion.

Cases 2, 5, and 8 show very marked gains in weight with only a trace of hydrochloric acid in the gastric juice. Case 2 is especially interesting. This infant was a typical example of infantile atrophy. Some idea of his emaciation may be gained by comparing his age and weight (6 months old and weighing a little over 8 pounds). He had been under observation for seven and a half weeks and had been fed on modifications of cows' milk during this time. His weight, when first observed, was 3,990 gm. For three weeks he was given 960 gm. a day of a whey-and-cream mixture. He lost weight and did not digest the food well. He was given a modification of milk without any whey for one week without any improvement. He was put back on the cream-and-whey mixture for two weeks, and on and after December 4th he was given 840 c.c. daily of a modification of milk which contained approximately 3 per cent. of fat, 6 per cent. of milk-sugar and 1 per cent. of protein. His weight at this time was 3,860 gm. The hydrochloric acid was determined four times between December 14th and 21st, and showed only a trace of acid. He continued to take this food until December 21st. On December 21st, his weight was 3,730 gm. He was transferred to the Massachusetts Infants Asylum, where, through the kindness of Dr. E. C. Stowell, I was able to procure breast-milk for him. On December 29th, his weight was 3,720 gm. From December 21st to December 29th, he had been fed on a whey-and-cream mixture and very little breast-milk and had not gained in weight. On December 29th and for the following three weeks he was fed on breast-milk alone, of which he took on an average 960 gm. daily. On January 5th he had gained 320 gm.; on January 12th, 285 gm.; and on January 19th, 180 gm., making a total of 785 gm. in three weeks (approximately 1¾ pounds). We ascribe the diminished gain of the third week to a change in wet-nurses, because during the third week the daily quantity of milk taken was not so large. We were unable to continue the observation any longer, because of

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the scarcity of breast-milk, and so he was given some breast-milk, but mostly modified cows' milk. The infant responded to the change in the food by ceasing to gain weight. Two months later he had gained only 200 gm.

Three determinations of hydrochloric acid, made at intervals of a week during the three weeks in which the gain in weight was so marked, showed no increase of hydrochloric acid.

Two things are important in this observation; first, the ability to assimilate a suitable food and to gain in weight at once without requiring time for the impaired functions to become restored, the infant previously having proved his inability to digest and gain in weight on certain mixtures of cows' milk in which the percentages of fat, sugar and proteid approximated those of human milk; second, the fact that the infant was able to do this without the aid of much hydrochloric acid. So far as the second point is concerned, if a number of similar observations were found to coincide, it would tend to show that, whatever part the hydrochloric acid may play in producing atrophy, it is not, under certain conditions, essential to recovery.

Patient 5 was under observation for two weeks. During this time he was fed on a whey-and-cream mixture and gained 410 gm. (approximately 14 ounces). One determination of hydrochloric acid was made and only a trace found. This infant was 3 months old and weighed 3,940 gm. (approximately 8¾ pounds). He did not present so extreme a degree of emaciation as some of the others. This food appeared to be suitable for this infant for the limited time that he was under observation.

Patient 8 was an extremely emaciated infant. He was 8 months old and weighed only 4,275 gm. (approximately 9½ pounds). On four feedings each of breast-milk and a modification of cow's milk he gained a little over 20 ounces in eleven days (615 gm.). The only examination that was made of the gastric juice showed a mere trace of hydrochloric acid.

The ability to gain in weight with very little hydrochloric acid in the gastric juice may throw light on the two cases which I reported last year (see Table 1, Cases O and P). Both of these infants had been atrophic, but had made marked gains in weight during the six or eight weeks preceding the time that the gastric contents were obtained. A single determination of hydrochloric acid made in each case showed it to be very much diminished.

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SUMMARY OF RESULTS.

1. The Sjöquist method rarely showed more than a trace of hydrochloric acid in the stomach contents from atrophic infants fed on cows' milk mixtures.

2. Out of twenty-one determinations made of hydrochloric acid in the stomach contents obtained from eight infants, ten showed a percentage of hydrochloric acid between 0.01 and 0.05, and eleven showed a percentage of hydrochloric acid between 0.002 and 0.009. These results correspond fairly well with eight determinations made last year (see Table 1) on the stomach contents from eight atrophic infants, of which seven showed percentages of hydrochloric acid between 0.01 and 0.041; and one showed a percentage of 0.07 hydrochloric acid.

The stomach contents from nine well-nourished infants showed a diminished amount of hydrochloric acid. The diminution of hydrochloric acid corresponded closely to that found in about one-half of the determinations made on the above-mentioned atrophic infants. Out of twelve determinations of hydrochloric acid in the stomach contents obtained from nine infants, one showed 0.12 per cent., one showed 0.09 per cent., nine showed percentages between 0.01 and 0.05; and one showed 0.005 per cent. hydrochloric acid. Thus the diminution of hydrochloric acid, although it was marked, was never so extreme as in one-half of the determinations obtained from atrophic infants. Repeated examinations could not be made in the normal cases and so the constancy of these results is problematical.

These results do not correspond well with those obtained last year from well-nourished infants (see Table 1) in which the percentages of hydrochloric acid were much higher.

3. From this series of observations it is not possible to show any definite or constant relation between the quantity of stomach contents obtained and the percentage of hydrochloric acid present. This statement applies equally well to any relations that may exist between the character of the food and the production of hydrochloric acid.

4. Examinations of the stomach contents obtained from the same atrophic infants at different periods of time after eating, and without any change in the diet, rarely showed any marked variations in the quantity of hydrochloric acid present.

5. The results of these observations do not prove that a

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marked diminution of hydrochloric acid in the gastric juice is an essential factor in the production of infantile atrophy, although they do show that this diminution is constant. On the other hand, considerable evidence is afforded by several of the observations that it is possible for atrophic infants to gain very rapidly in weight and yet produce very little hydrochloric acid in the gastric juice. This appears to show that the quantity of hydrochloric acid in the gastric juice is not an important factor in recovery from infantile atrophy.

Briefly, then, these observations show the following facts:

1. A constant and marked diminution of hydrochloric acid in the gastric contents from atrophic infants.

2. Great variations in the quantity of hydrochloric acid in the gastric contents from well-nourished infants.

3. In this series of cases of infantile atrophy, there was little, if any, increase of hydrochloric acid in the gastric juice as the result of prolonged digestion.

A few words in conclusion about the method employed in these observations. These observations were not made for the purpose of comparing normal and atrophic infants, nor was it intended to show the exact quantity of hydrochloric acid present in the gastric juice from atrophic infants. The sole purpose was to find out whether hydrochloric acid was constantly and markedly diminished in the gastric juice in cases of infantile atrophy. Any criticism that may be made as to the accuracy of the Sjöquist method when milk is the diet is irrelevant when applied to my results, because only such a constant and marked difference between the normal and the atrophic infant as to place the result beyond the range of error in the method could be of value so far as my hypothesis was concerned.

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THE NUTRITION OF THE FEEBLE INFANT.

BY HENRY DWIGHT CHAPIN, M.D.,
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The proper nourishment of feeble infants is a problem that taxes the resources of the most skillful pediatrist. Digestion and assimilation may be poor either by inheritance or from faulty feeding, and when, from any cause, the general vitality of the infant is lowered, the digestive tract is usually the first to suffer and the last to recover tone. Whether the infant becomes feeble from errors in diet and hygiene, from illness or from poor inheritance, improved diet and hygiene form the only successful treatment. Drugs are of little or no avail. If these patients are not relieved, sooner or later some form of atrophy or marasmus will result—a condition in which a chronic digestive weakness is the principal evidence of the depressing symptom-complex. The large general death-rate of early infancy can be checked only by the most careful attention to the diet and care of this class of feeble infants. It must be confessed that thus far our results have not been very brilliant. The object of this paper is to discuss some of the factors that enter into this problem, with the idea of suggesting the lines along which improvement may take place.

The structure and function of the digestive tract, the form and quantity of the food elements that are given, especially in relation to the assimilative power of the individual, and the most improved hygiene must all enter into such a discussion. With reference to the ingestion of food, the stomach will naturally call for the first consideration.

I. *The Motility of the Stomach.*—Our knowledge of the general capacity and function of the stomach in early life has been fairly well worked out. In normal infants, during health, no great difficulty is encountered by competent feeders in reference to the kind and quantity of the food and the intervals of its administration. In feeble and badly nourished infants, however, the weak action of the stomach, especially in its muscular power, is the source of much trouble. The weakness and atrophy of the general musculature is exemplified in a marked degree in the motility of the stomach. At best, the musculature of the stomach

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and intestines is not highly developed during the first months of life, and in feeble infants, in whom the general muscular tone is below par, the digestive tract suffers very markedly from this cause. This will doubtless throw some light on those cases in which during life serious digestive symptoms have been noted, but after death little pathologic change has been observed as far as the mucous membranes are concerned.

With this condition in mind, a series of 21 cases was carefully studied with regard to gastric motility, as in the case of the feeble infant it is important to make certain that the intervals of feeding are not too frequent. The ages of the patients were as follows: Two weeks, 1; three weeks, 1; six weeks, 1; two months, 1; three months, 6; four months, 4; five months, 2; six months, 1; seven months, 1; twelve months, 3. These infants were taken from the wards of the hospital and represent all grades of malnutrition from beginning cases to those in the last stages of atrophy. We may generally classify the cases as 13 of marked feebleness, 3 cases of malnutrition in a milder form and 5 cases just at the beginning of faulty nutrition. It was, however, impossible to form any definite conclusions as to gastric motility based on the age of the child from these cases. The older children were brought to the hospital either suffering from some grave general disease, or because they had failed to gain weight and were becoming atrophic. They thus formed poor material from which to determine normal gastric motility.

Of 19 patients that were examined at the end of three hours, only 6 could be shown to have approximately empty stomachs, and these six, with one exception, were holding their own, and in no case could free hydrochloric acid be determined. In 13 out of the 19 cases it was absolutely demonstrated that there was a considerable residual amount of food still remaining in the stomach. In all these cases there was a positive Fehling test, and curds and whey, either singly or together, were present. It is interesting to note that butyric acid fermentation was marked in 6 of the cases, and in 2 of the cases in which the stomach was apparently empty there was a faint trace of butyric acid. In 5 of the 7 patients examined at the end of three and one-half or four hours, the stomachs were surely empty. Of the remaining 2 cases, 1 showed many curds at the end of four and one-half hours, while the other showed only a few small curds. These last 2 patients were losing weight rapidly and the butyric acid fermentation was marked.

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In only 2 of the 17 patients examined at the end of two or two and one-half hours, were the stomachs empty. In the other 15 cases a positive Fehling test was obtained with one exception, and in four there was a positive Gram test. In 13 of these cases butyric acid fermentation could easily be demonstrated. Large tough curds, or many small curds with whey, were present in all of these cases. In considering the above figures, it should be remembered that few, if any, of the children examined were entirely normal, and that deductions from these results have a value only as a study in the nourishment of the feeble child.

There can be little doubt that, in a large majority of cases, the feeble infant with digestive disturbances has been fed at too frequent intervals. Too often a second feeding is given before the previous meal has passed through the pylorus, with the result that each successive feeding has been contaminated. The process of butyric acid fermentation has thereby been set up and kept in operation until, at last, a condition analogous to that of myasthenia gastrica is present. In none of these cases could any great disturbance in acid secretions be made out. The condition of the enzymes was not carefully investigated, but, as it is a well-known fact that the enzymes are not affected until long after the acid secretions have become greatly diminished or disappeared, it may be taken for granted that the enzymes were present in a normal state. While free hydrochloric acid could be demonstrated in only 6 cases, combined hydrochloric acid was invariably present in fairly large quantities. The average amount was about 30, the lowest being 12, and the highest 48. In nearly every case in which the stomach was not empty, fluid was present, and in a few cases the dilatation was marked. This state of affairs serves to emphasize the fact that in feeble infants gastric motility is greatly decreased, and that whatever may be the case in the normal healthy child, in these atrophic infants it is often necessary to increase the interval between the feedings.

According to Tobler and Bogen and Heubner, milk passes through the pylorus in the case of breast-fed infants in about two hours, and in those taking cow's milk in about three hours. Tobler has further shown, as a result of experiments carried on in animals with variously colored milks, that each subsequent feeding forms a sort of layer about the earlier one, and thus the food first ingested may be kept in the stomach indefinitely if the feedings are given at too frequent intervals. Granting the truth

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of these observations, it can be seen at once that it is of the utmost importance to have the meals properly spaced, as otherwise there remains a residuum of food in the stomach which contaminates each successive addition of food. This constantly recurring contamination and overburdening of the stomach results in fermentation, and in time gastric dilatation with a loss of tone of the muscular coats of the stomach may ensue. This explains how a chronic digestive disturbance may lead to the condition known as atrophy.

It is interesting to trace the analogy between this state in the infant and the condition known as myasthenia gastrica in the adult. Myasthenia gastrica is characterized by a loss of tone in the muscular coats of the stomach, as a result of which there is a moderate dilatation. Its diagnosis depends on the finding of fluid in the stomach at the end of two hours or more, with a normal gastric secretion. Among the causes bringing about this condition may be mentioned the general debility following any disease and the habitual ingestion of excessive amounts of food. An important element in its treatment is to make certain that the stomach is never overloaded. In atrophic infants these conditions are reproduced, and while it cannot be claimed that they are exactly parallel there is a good deal of similarity. Thus in atrophic infants a practically normal gastric secretion may be found with a marked loss of motility if our previous schedule of three-hour feedings be accepted as approximately correct. With few exceptions, food and fluid remain in the stomach of these infants well over two hours—in most cases three hours or more—and as a consequence of this stagnation butyric acid fermentation begins and is passed along from one feeding to the next. Hence it should be expected, as in the myasthenia gastrica of the adult, that there should be a marked improvement in many of the symptoms if care is taken to make certain that the stomach is entirely empty for a short period before a future feeding. It is not only desirable that any fermenting material lying in the stomach be passed forward into the intestines to avoid contamination of an incoming meal, but there should also be an interval during which the stomach contents shall be strongly acid, so that the growth of harmful bacteria may be inhibited.

II. *The Form and Quality of the Food Elements.*—When percentage feeding was first developed, it was assumed that protein, fat, carbohydrates and mineral matter were well-defined

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entities, and the basis of teaching was that scientific feeding consisted in merely changing the quantities of these ingredients in the food mixtures. Within the past few years an entirely different viewpoint has been taken, principally as the result of an increase in general knowledge of the subject of nutrition. "Protein," "fat," "carbohydrates" and "mineral matter" are now recognized as being generic terms and just about as definite as the terms "wood" or "stone." There may be few who realize that at least twelve different forms of protein, five different forms of carbohydrates and two different forms of fats are used successfully in infant-feeding and the digestive properties of these food elements are modified by the combinations in which they are offered.

To be most successful as a feeder one must be familiar with the uses and fields of these different forms of the food elements, and must also realize that there is no fixed and absolute rule to follow in combining them. There are many feeble infants who seem to thrive when the food contains a high percentage of carbohydrates, while others do better if the carbohydrates are kept low and the quantity of protein is raised. Occasionally raising the fats will improve nutrition and this may appear to be what the infant needs and can digest; but most feeble infants seem to have difficulty when the fats are too high. Milk mixtures containing much cream pass more slowly through the pylorus than those low in their percentage of fat and higher in protein and carbohydrates, according to Tobler and Bogen. Many will remember that only a few years ago high fat in the food was looked on by many as the thing to be especially desired, but now low fats seem to have the field. There can be no universal rule with reference to the fats, however, any more than to any other of the food elements.

From the standpoint of nutrition infants need liberal quantities of protein for tissue formation, and enough fats and carbohydrates to supply heat and energy value or calories. But in the management of feeble infants the question of what is theoretically correct must be subordinated to what will practically work, or, in other words, what the infant will take and assimilate.

In some instances a very small quantity of protein, perhaps 0.5 per cent., is all that the young infant can take when the protein is in the form of the natural casein of milk. If the form of the casein is changed by the action of an acid, as in buttermilk feeding, in which the casein is precipitated in a finely divided

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state, as much as 1 or 2 per cent. of the casein may be digested easily. Lime-water, sodium bicarbonate, sodium citrate and other chemical additions for modifying the casein have been used with milk, and all produce good results in some cases. No one method has succeeded in all instances, and it is not likely that any will ever be discovered that will be universally applicable, for the reason that infants are not all alike and conditions are not always the same. If oat gruel, or soy-bean gruel, with its very high protein content, is employed, several times as much protein may often be utilized as if the unmodified protein of cow's milk is used. These facts point out plainly that changing the form of the protein has frequently a very important place in the management of infants.

The changing of the form of the carbohydrates also frequently shows a marked effect in the progress of the infant. The carbohydrates used in infant-feeding are starch, dextrin, maltose, cane-sugar and milk-sugar, and it is often surprising what marked improvement will follow a change in the carbohydrates employed.

It has generally been thought that infants cannot take more than 7 per cent. of carbohydrates, but in Keller's malt soup, which has been advocated for atrophic infants, the carbohydrates, composed of starch, dextrin, maltose and milk-sugar amount to about 13 per cent., or almost double the accepted maximum quantity. The percentage of fat in the malt soup is about 1 per cent. and that of protein about 1.7 per cent., and each ounce yields about 22 calories, or almost exactly the same as 1 ounce of good breast-milk. In many instances Keller's malt soup has given good results, while in many others it has signally failed, like every other mixture. Its use proves only one thing; there are some patients who seem to thrive especially well when the fats are low and the carbohydrates high and consisting of a mixture of several forms.

I have long advocated the use of dextrinized gruels for this reason and for the practical results in many cases of difficult digestion. As usually employed, the carbohydrates exist in these gruels in the forms of soluble starch, dextrin and maltose in varying proportions, according to the degree to which the digestive change has been carried. Some atrophic patients, however, do better when the product is largely maltose. This can be attained by allowing an active diastase to remain in the gruel for a longer time at a comparatively low temperature.

The general method to be employed in feeding in difficult cases

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will have to be determined, to a certain extent, by experiment with the individual infant. If this fact is once firmly fixed in mind much progress will have been made, as time spent in looking for a universally applicable food will then be used in learning how to make up mixtures of equal nutritive value in which the various food elements may be high or low in quantity and in a great variety of forms. Where the physician now has one or two formulas to draw on he may thus have many, and as he learns to use them he will find that the difference between success and failure often lies more in changing the form and even the flavor of the food than in a profound knowledge of calculating theoretically indicated mixtures. This is particularly the case in dealing with the difficult digestive problem of feeble infants.

III. *Finkelstein's Method of Feeding.*—An interesting and somewhat unique contribution as to the cause and relief of nutritive disturbances in infancy has recently been made by Finkelstein, of the Berlin Kinderasyl. Instead of the fat or protein being the usual cause of trouble, most digestive disturbance is referred by him to the sugar of milk. He confirms* an earlier report of clinical observations at the Berlin Kinderasyl in reference to the pathogenesis of severe nutritional disturbances in the nursling. He there found that the ingredient in the food especially harmful to the sick infant was the sugar, and that in certain cases even the natural milk-sugar of cow's milk might cause the severest symptoms. He was thus led to hope that a method of nourishment based on a decrease in the milk-sugar would lead to cures that would be impossible with the ordinary methods. The report of improvement following this kind of diet has proved the correctness of the hypothesis, and Finkelstein is convinced that a step in advance in the treatment of the nutritional disturbances of poorly nourished infants has been made. Not only may the lighter forms of nutritional disorders be quickly and certainly improved, but patients can be cured that our previous experience has shown could be saved only by breast-milk. The earlier view of the harmfulness of casein is not confirmed, and the opinion is advanced that casein in the intestinal canal is directly beneficial, as the intensity of the pathologic sugar fermentation, with the ensuing dyspeptic symptoms, is lessened by increasing the amount of casein. A lowering of the sugar increases the tolerance of the intestines for fats, and hence will allow an increase in the amount

* Finkelstein: *Monatschr. f. Kinderh.*, April, 1909.

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of fats which in the former mixtures, rich in milk-sugar, only work harm. The principles of the new method of nourishment are summarized as follows: (1) The cutting down of the amount of milk-sugar and of the salts; (2) in place of this casein is added and a not inconsiderable amount of fat; (3) a further bettering of the condition is sought by substituting other forms of carbohydrates for the milk-sugar, which tends to an increased tolerance.

The food is prepared by separating the casein and fat from one liter of milk. The curds are pushed through a sieve to break them up and then added to half a liter of water. Half a liter of buttermilk without sugar is then added to the mixture. One liter of this food contains the whey and sugar from half a liter of milk, the casein from one and a half liters and the fat from one liter. A small amount of fat and casein are lost in its preparation, but the following are estimated as the percentage ingredients —protein, 4.5 per cent.; fats, 2 to 3.5 per cent.; sugars, 1.5 per cent.; salts, 0.3 per cent.

TABULATION OF RESULTS FROM FINKELSTEIN'S FEEDINGS.

Case	Age, Weeks	Ordinary Feedings			Finkelstein's Feedings			Condition at the Start of Finkelstein's Feeding	Final Condition		
		Days	Gain Oz.	Loss Oz.	Days	Gain Oz.	Loss Oz.			Gain Oz.	Loss Oz.
1	3	..	Stationary		17	8	....	Good.	Improved.	8	..
2	3	..	..	....	21	5	....	Good.	Improved.	5	..
3	4	8	..	2	29	..	7	Fair.	Death.	..	9
4	6	30	..	12	6	..	7	Marked atrophy.	Death.	..	17
5	6	10	..	7	6	1	....	Beginning atrophy.	Death.	..	6
6	8	9	..	10 ½	32	15	....	Beginning atrophy.	Improved.	5	..
7	8	32	..	10	27	5	....	Fair.	Death.	..	5
8	8	15	..	10	36	..	23	Good.	Death.	..	28
9	13	6	..	4	14	15	....	Fair.	Improved.	7	..
10	13	..	..	....	22	..	1	Good.	Stationary.	..	1
11	13	37	..	25	27	19	....	Atrophic.	Improved.	7	..
12	13	..	..	....	22	..	30	Beginning atrophy.	Death.	..	30
13	13	23	..	17	3	..	3	Atrophic.	Death.	..	20
14	13	13	..	19 ½	2	..	4 ½	Marked atrophy.	Death.	..	24
15	13	16	..	16 ½	6	..	6	Moderate atrophy.	Death.	..	22½
16	13	6	..	10	7	..	10	Atrophic.	Death.	..	20
17	13	15	..	3	11	..	18	Good.	Death.	..	20
18	17	27	..	19	51	..	8	Atrophic.	Death.	..	27
19	21	3	4	....	31	2	....	Good.	Improved.	5	..
20	26	13	..	7 ½	6	..	4	Marked atrophy.	Death.	..	11½

This method of feeding was tried at the Babies' Wards of the New York Post-Graduate Hospital in a series of 20 cases. The infants, with a few exceptions, were not doing well on the ordinary food when they were changed to the new method. They were all hospital patients and presented the usual difficulties of infants previously badly fed and cared for, and finally sent to an institution for treatment. When the change was made the infants were

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fed every four hours, day and night, and the food was usually given in lesser bulk than in the previous feedings. Water was given freely between the feedings. The stools generally became gray and homogeneous with a putty-like consistency and dry appearance. There was usually then a tendency to constipation. The results are given in the tabulation above.

A study of this table will show that the results in this series of cases were not very promising. Before starting the new feeding the ordinary percentage method, with and without gruel diluents, had been employed with poor results. At the beginning of the new feeding the infants, as to their nutritional condition, may be classified as follows: In good condition, 6; in fair condition, but a little below normal, 3; beginning atrophy, 4; with atrophy, 3; with marked atrophy, 4. Twelve patients died; 8 of these began as atrophic infants and 4 started in good condition when the diet was instituted. Three patients, which began as atrophic infants, increased in weight, and 5 increased somewhat in weight who began in good condition. Altogether, 8 patients gained slightly in weight and 12 lost in weight on the Finkelstein feeding.

It seems to me that when benefits follow this method of feeding the results are due more to the form in which the protein is given, the casein being in a very finely divided state, than to the lessened amount of sugar in the mixture. The food appears in some cases to check the rapidity of atrophy temporarily and hence may have a restricted usefulness. Judging from the character of the stools it may also act well in certain forms of diarrhea.

IV. *Hygienic Management.*—The hygienic surroundings have a most important effect on the nutrition of the feeble infant. If the environment is faulty the best care and feeding will usually prove ineffectual. These patients require an altered environment that will furnish plenty of fresh air, good general hygiene and individual care. For this reason they never do well in institutions, no matter how carefully and scientifically they are there fed. They cannot assimilate the best of food without plenty of good air to assist in its oxidation; oxygen is as necessary a food for them as protein or fat. It is only individual housing and care, with constant oversight, that can accomplish good results. Even an ignorant but kindly woman in a home can often get better results than a trained nurse in a hospital with a series of cases to look

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after and a stated routine to enforce. This is especially true in charitable work, where relief of feeble infants can be much better accomplished along the lines of family life, with individual supervision, instead of the collective life with institutional methods.

Acting on this idea, the Speedwell Society was started at Morristown, N. J., in 1902, and I have ever since boarded out my atrophic infants there under the supervision of a doctor and trained nurse, who watch and treat the cases under the care of the various foster-mothers. The results have been better than with any other method of treating this class of cases. Thus, among 121 infants under three months, 45 died; 95, from three to six months, had 29 deaths; 83, from six to twelve months, had 21 deaths, and 85 infants, from 1 to 2 years, had only 8 deaths. These infants were all poorly nourished at the start, from bad hygienic surroundings, with various degrees of digestive disturbance from faulty feeding on the bottle, and stationary or losing weight. Although all had to be kept on bottle-feeding, a good proportion were not only saved, but were restored to fair and even vigorous vitality. Under the old institutional methods nearly all would have died.

I would express my thanks to Dr. Pease and Dr. Leopold of my staff. to the former for undertaking the work on the motility of the stomach, and the latter for applying the technic in the Finkelstein trials.

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SOME OBSERVATIONS ON THE EFFECT OF FAT IN THE DIET OF INFANTS.*

BY JOHN HOWLAND, M.D.,
AND
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This report is part of a piece of work which we have undertaken in regard to the metabolic effects produced by the ingestion of varying amounts of fat in the diet of infants. It was undertaken with the desire of reconciling the conflicting chemical testimony which has been advanced chiefly by German investigators and the clinical testimony of some who habitually feed infants large quantities of fat. One receives the impression from the work of the former that fat feeding pushed beyond a very moderate point is detrimental to the health of a child, and that this injurious effect is manifested promptly by chemical changes in the excreta. On the other hand, we see as the result of almost every-day experience that some children may take large quantities of fat over long periods of time with apparent benefit and without the least indication, so far as their general health is concerned, of any detrimental effect.

Keller, and later Czerny and Keller, showed that children with gastroenteritis excreted a relatively large amount of their urinary nitrogen in the form of ammonia, and that the ammonia was promptly increased by the addition of fat to the diet. The explanation for this was sought in the absorption of acids from the alimentary canal or in the formation of abnormal acids in the intermediary metabolism. A search for such acids, however, failed to reveal them; those found in the urine differed in no wise from the normal. Steinitz in 1902 was able to increase the ammonia excretion in a normal and two abnormal infants by the feeding of fat in excess, and he showed that when the ammonia percentage rose there was a loss of potassium and sodium by the intestines. The ammonia increase was a compensatory process to neutralize the normal urinary acids which had an insufficient amount of sodium and potassium to bind them on account of the loss of these in the stools. All of the infants were under four months of age, and the amount of ingested

* From the Children's Wards, First Medical Division of Bellevue Hospital and the Loomis Laboratory.

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fat which produced the abnormal ammonia excretion was about 22 grams a day. This was in a food containing 4 to 5 per cent. of fat. Steinitz, while not directly making the statement, gives the impression that it is his belief that fat in this quantity will regularly produce these abnormal changes. More recently it has been shown by L. F. Meyer, Bahrdt and others that sufficient fat may be ingested to cause the large, white, dry "Fettseifenstühle" and yet no loss of alkali by the bowel take place and no increase of ammonia in the urine. Bahrdt has insisted that with such "Fettseifenstühle" there is no loss of sodium and potassium, but only the excretion of a relatively large amount of calcium, not sufficient, however, to cause a minus balance, and that the loss of alkali only takes place with the loose, watery, dyspeptic stools in which there is an increased nitrogen and mineral loss due to the greater secretion from the wall of the intestine. By all, however, the danger of a large amount of fat is constantly reiterated, it being insisted that when sodium and potassium are lost in large amount with the so-called dyspeptic stools a demineralization of the organism occurs, and with the so-called "Fettseifenstühle" the excretion of calcium in excess interferes with nutrition, causes chronic disturbances and favors the development of rickets. Clinical experience, as stated before, has shown that some children take for months a very much larger amount of fat in their diet than Steinitz believed dangerous, that they may gain weight and strength and manifest no nutritional disturbances or evidences of rickets.

We have, therefore, investigated a series of cases through many periods on different diets, making complete analyses of the food, urine and feces. At the present time we are in a position to report upon a few points in connection with the work, and, as they have a clinical interest, we present them in their unfinished condition. We have selected for presentation the results obtained on three infants who were put in a metabolism apparatus of our devising, which has the advantage of allowing a greater freedom of movement than others described. The infants apparently suffered not the slightest inconvenience, but were rather more contented, perhaps, on account of the increased attention which they received.

A diet was given containing a fairly constant amount of nitrogen; the amount of fat and carbohydrate was varied. On a fat-free diet it is impossible to furnish all the calories one would

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wish by sugar, as it almost invariably causes vomiting, and the energy supply on the fat-free periods must therefore be comparatively low (85-90 calories per kg.).

On the high fat diet the caloric value of the food rises very rapidly, and on the highest fat diets it reached 159 calories per kg. For these reasons the energy quotient could not be kept constant.

The protein given represented .6 gram of nitrogen per kilo.

CASE I. Infant of ten months, who at the beginning of the experiment in December weighed 11 pounds (5 kg.) and was in poor condition as a result of gastroenteritis and double otitis media the summer before. This boy has been studied through eleven periods of two to four days each. We began with a practically fat-free diet and increased his fat gradually until he was taking 76 gm. of fat a day (a food containing over 7 per cent. fat). We kept him upon this high fat diet for two and a half months and then reduced him to a fat-free diet again. When on the fat-free diet, both at the beginning and close of the experiment, his stools were brownish, frequent and thin. As the fat was increased the stools became lighter and dryer, and on the high fat diet were white with a tinge of green, dry and very large.

As will be seen by the chart the nitrogen intake was fairly constant, while the calories were increased from 360 (68.5 per kg.) to 934 (159 per kg.), and then were reduced again to 605, or 86.5 per kg. The chart also shows, except on the first low fat period, when the loss was probably due to a deficient energy supply, a nitrogen retention throughout which was greater with the increase in fat and is to be attributed to the much higher caloric value of his food.

Ammonia.—Throughout the experiment the ammonia percentage is at the upper limit of what may be termed normal. With the increase in the fat of the diet there is a distinct rise in the percentage till at Period 4 it reached 13.6 per cent. But with the continuance and even increase of the fat in the diet it again fell to 8-9 per cent. This decline in ammonia percentage at least suggests that the child may accommodate himself to the utilization of what was before sufficient to give evidence of disturbed metabolism. This diet was higher in fat and was continued for a much greater length of time (seventy-six days) than that in any experiment reported in the literature, and yet during all this time the child remained in excellent physical condition and gained weight rapidly. He is now a fat, robust child, shows no trace

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of rickets, has good color and has gained 5 pounds in four months. It must be considered that this child did very well throughout the whole experiment.

CASE II. W. L. Six months. An undernourished child who some weeks before the experiment was admitted to the hospital with otitis and an ischiorectal abscess, which healed promptly under treatment. He was carried through four periods, the first three of three days each, the last of only twenty-four hours, on account of the gastric disturbance. The fat was practically absent in Period 1 (1 gm.) and was increased until it reached 66 gm. in Period 4 (8.4 per cent.).

CASE I. J. T., TEN MONTHS OLD.

	Food				Urine			
	N	Fat	Carbo-hydrate	Cal. per Kg.	N	%NH3	Fecal N	N balance
Period 1*...	2.87	2.59	66.4	68.5	2.44	9.8	.52	— .10
" 2 ...	3.6	18.	46.5	87.	2.91	7.6	.32	+ .37
" 3 ...	3.6	42.	26.5	113.	2.6	9.7	.33	+ .67
" 4 ...	4.18	59.	30.	141.	2.8	13.6	.23	+1.14
" 5 ...	4.0	76.	30.	159.	2.8	9.6	.30	+ .86
" 6 ...	3.8	1.3	121.	86.	2.7	7.	—	—

CASE II. W. L., SIX MONTHS OLD.

	Food				Urine			
	N	Fat	Carbo-hydrate	Cal. per Kg.	N	%NH3	Fecal N	N balance
Period 1 ...	3.5	1.2	90.	85.	2.8	12.	.49	+ .23
" 2 ...	3.7	21.	46.	86.	2.5	8.4	.32	+ .88
" 3 ...	3.6	57.	34.	136.	2.8	14.	.26	+ .59
" 4 ...	2.7	66.	28.	141.	2.4	21.	.21	+ .19

CASE III. T. S., FIVE MONTHS OLD.

	Food				Urine			
	N	Fat	Carbo-hydrate	Cal. per Kg.	N	%NH3	Fecal N	N balance
Period 1 ...	2.2	1.4	55.	82.	1.8	11.5	.32	+ .16
" 2 ...	2.3	15.	34.	96.	1.3	13.2	.29	+ .66
" 3 ...	2.3	31.	21.	124.	1.3	21.	.26	+ .77

* The figures for each period are calculated in terms of 24 hours.

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The feces of the first period were watery, frequent, yellowish or brownish and of foul odor, which is characteristic with a diet very rich in carbohydrate. As the fat intake was increased the stools became formed, lighter in color and eventually almost white and rather dry. The nitrogen balance was always positive, and the retention was greatest with a moderate amount of fat in a diet containing only 85 calories per kg. It was less on the very high fat diets, which contained 136 and 141 calories per kg. respectively.

Ammonia.—The ammonia output was relatively high throughout the entire experiment. It was lowest on the moderate fat diet and increased enormously during the last period with the very high fat intake until it reached 21 per cent. of the total urinary nitrogen.

It is worthy of emphasis that this very high percentage of ammonia occurred with the white dry stools, with which it is claimed there is no loss of sodium and potassium.

On the medium fat diet this child did best. When the fat was increased he still gained in weight, but both chemical examination and clinical observation suggested that he was disturbed, and during the last period, with the very high fat intake, he refused one of his feedings entirely and vomited part of another. For this reason the period was stopped at the end of twenty-four hours. After the experiment the fat was reduced to a low point. His appetite remained poor for several days, but thereafter his digestion improved and he is now gaining satisfactorily.

CASE III. T. S., a very small malnourished infant of five months, was admitted to the hospital on account of his poor condition. Before the experiment his digestion had improved but there was no gain in weight.

The investigation lasted three weeks, during which time there were three periods in which he was on a fat-free, a medium and a high fat diet, the last being a food with 3 per cent. fat, representing 124 calories per kg. The stools in all three periods were fluid, rather copious and very foul smelling; the only change was one of color, the high fat stools being whitish, the others yellowish. There was a slight loss of weight—.1 kg. during the experiment.

Nitrogen.—Despite the obvious disturbances, chemically and

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clinically, the nitrogen retention was continuous and was best on the high fat diet.

Ammonia.—The ammonia nitrogen was relatively high throughout the experiment, and increased with the increase in the fat of the food, reaching 21 per cent. in the last period. At no time during the experiment did the child do well. The last period was of only twenty-four hours because of an increase in the frequency of stools and a general poor condition. Partial feeding with breast milk improved him greatly and he is now doing well on a low fat mixture.

These 3 cases represent different reactions to the high fat feeding. *The first child* took a very large amount of fat over a prolonged period of time with no permanent urinary change to indicate any bad effect and no clinical evidence of anything but benefit. This might be attributed to the fact that he was a little older (ten months) and that the fat increase was more gradual. But, on the other hand, the duration of the experiment gave ample opportunity for the development of any disturbance.

The second child took a moderate fat diet well, but with an excessive amount of fat he gave clinical and chemical evidence of decided disturbance.

The last child did not do well with any diet and was particularly upset by a moderate fat intake. With the last two children the recognition of the disturbance from the fat was as prompt clinically as chemically. Our results may be taken as corroborating in part the clinical experience that infants exhibiting the large white stools characteristic of a rich fat diet may do well, as shown by Case I., or with stools of the same character may give evidences of disturbance, as Case II. indicates. They show also the danger of drawing too definite conclusions from a limited number of observations, and they throw doubt upon the deductions of some of the German experimenters that high fat feeding invariably produces changes that must sooner or later be detrimental to the condition of the infant. We would not be understood in the least as advising the use of excessive quantities of fat. We believe that chemical testimony and clinical experience teach that it is, in many instances, badly borne and is a dangerous procedure. It is nevertheless true that the individual tolerance of some infants is so great, as our Case I. indicates, that a gross variation from a normal diet may produce nothing abnormal.

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In this connection it is interesting to see that two of the chemical variations emphasized as an indication of the bad effect of fat, viz., an increase of urinary ammonia and fecal nitrogen, are present when the diet is as near fat free as can be made. A consideration of the reasons for these variations on this diet would take us too far afield.

The best diet is undoubtedly one containing fat and sugar, both in moderation: the absolute limit for either of them cannot be set by any series of experiments on account of the great individual variation of infants; the limits beyond which it is dangerous to go can most satisfactorily be reached as the result of a great number of careful clinical observations.

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DISCUSSION OF THE PAPERS ON DIETETICS.*

DR. MEARA.—These papers involve such detailed work that they can scarcely be discussed without careful consideration and what I have to say is merely in the form of a question.

I would like to ask Dr. Wentworth if he took into consideration the relation of the fat to the quantity of hydrochloric acid obtained? Were the contents taken from the stomach as a whole examined as to hydrochloric acid. The same idea would obtain with reference to the quantity obtained at various times.

DR. COWIE spoke of the correlation between pyloric action and the degree of acidity. Might not the spasm be attributed to the effect of the acid on the duodenal side rather than on the gastric side?

DR. CHAPIN'S remarks as to the frequency of feedings are very important. Tobler has told us that in one experiment where the feedings had been at intervals of one and a half and two hours milk was obtained from the core of the curd unchanged after nine hours, but containing 16,500,000 bacteria to the c.c. instead of 10,000 per c.c. as ingested. There is a true gastric lipase in these infants' stomachs which may cause a splitting up of the fatty acids. In the test tube lipase from the infant's stomach has split up in twenty-four hours 20 per cent. of an egg-yolk emulsion with which it was in contact. The result of too frequent feedings may be not only retention of food products but an increased bacterial content.

As to Finkelstein's method, I had the impression that the earlier evidence of loss of balance was due to the fat and not to the sugar, and that some of his best results were in an increase of certain sugars. These experiments would have reference to cases of febrile reaction or alimentary intoxication rather than to disturbance of balance.

DR. WENTWORTH.—I would like to ask Dr. Veeder if the periods were continuous or not—three day periods? And if so, if one period ran into the other? It is difficult to estimate the effect on digestion where one varies the fat so much as that without proper intervals between the observation.

I would like to ask what the percentage of fat per gram of dried feces is and also what percentage of this was found as soaps. So far as my experience goes 90 or more per cent. of the fat excreted in the feces is in the form of soaps.

DR. CHAPIN.—In reference to Dr. Ladd's paper, so far as

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* These papers are by the following:

Dr. Herman,	see page 17.	Dr. Wentworth,	see page 111.
Dr. Ladd,	" " 26.	Dr. Chapin,	" " 120.
Drs. Cowie and Lyon,	" " 91.	Drs. Howland and Cooke,	" " 130.

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the incubator and padded crib are concerned, I prefer the padded crib. As the result of experimenting with 200 premature infants I found that the best incubator is not as good as the crib. These infants are unusually susceptible to lack of fresh air and I would rather have a varying temperature with pure air than the steady temperature of the incubator with impure air. If the incubator is used, it should have connection with outside air. These incubator babies should always be fed on breast milk. I have never saved a premature infant without breast milk. I have a wet nurse on call for such cases, as I feel that I cannot save these infants otherwise.

Dr. Abt.—The Society is particularly fortunate in having an opportunity to listen to this valuable series of papers. I have been following for some time this chemical research work and have been somewhat impressed with the discrepancy in results and with the various interpretations. We all feel the same way; that it is pioneer work; blazing the way; and that we will get important results ultimately. I think, however, that the dogmatic methods used in some quarters in making interpretations and attempting to make clinical deductions based on chemical results, thus far obtained, is somewhat premature. For instance, it is common in Germany to teach that all of the intestinal disturbances are due to defects in the chemistry of the fat, sugar or proteids. I think that this is a little premature. I am impressed with the hypothesis of Pfaundler, that besides the chemistry of digestion there is a biology of digestion; that the specificity of the biology of the individual cell—the ultimate appropriation of the molecule and its combination with the cell, is of more importance perhaps than these chemical details.

Dr. Samuel L. Bernstein, Cleveland (visitor): In connection with Dr. Ladd's paper, I would simply corroborate what Dr. Chapin has said. The use of the electric heating pad and maternal feeding have been the means of saving a number of these premature infants in our institutions. The electric pad replaces the hot water bag very well.

Dr. Cowie.—In reply to Dr. Meara's question as to whether the acid acts on the pyloric or duodenal side, I am inclined to believe that it is on the duodenal side that the acid has to do with the retention of food in the stomach.

Dr. Wentworth.—I don't think I understand exactly what Dr. Meara means. The purpose of this work was to determine whether there is a constant and marked diminution of hydrochloric acid in the stomach contents from atrophic infants. The quantity obtained from the stomach was all that I could express by any method used. The entire amount was thoroughly mixed and a portion of it taken for analysis. Duplicate analyses were always made. The results showed that there was a diminution of hydrochloric acid in every case.

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DR. HEIMAN.—When we find that certain infants take as much as 200 grams of milk we consider that a curiosity. We are supposed to judge by the average child. These experiments show that these babies take a good deal more and it will probably suggest to us in the future to feed at longer intervals.

Leo, in 1888, showed that free hydrochloric acid acts as a bactericidal agent in the stomach.

DR. B. S. VEEDER, Philadelphia (guest): In regard to Dr. Wentworth's question, these experiments extended over seven or eight weeks. The child was first put on a formula for three or four days and then put upon the metabolic bed on the same formula.

As regards the percentage of fat in grams, the figures refer to total weight of fat. As a rule, the weight was about 50 per cent. The soaps are included in that.

DR. WENTWORTH.—You didn't test for the soaps separately?

DR. VEEDER.—No; we know what proportion of soaps there is however.

The bed we used was the one invented by Dr. Howland, and we found it better than any other that we have worked with. Our results do not show anything definite. It is only a preliminary report and we hope to go on and make a large number of such studies. It will take a large number before we can draw any definite conclusion.

DR. CHAPIN.—There were several steps in my paper that I did not have time to bring out. Dr. Meara spoke of the fat forming layers. I should like to read a paragraph from my paper bearing on this: "Granting the truth of these observations, it can at once be seen that it is of the utmost importance to have the meals properly spaced, as otherwise there remains a residuum of food in the stomach which contaminates each successive addition of food. This constantly recurring contamination and overburdening of the stomach results in fermentation and in time gastric dilatation with the loss in tone of the muscular coats of the stomach, thus explaining how a chronic digestive disturbance may lead to the condition known as atrophy."

In these 21 cases that is exactly what happened.

I am in sympathy with the remarks of Dr. Abt about studying these questions in detail and along the broad lines of biology. It seems to me to be the only way of studying infant feeding. The matter must be studied in relation to the broad question of digestion and assimilation.

DR. HOWLAND.—This biological question is largely hypothetical. It cannot be expressed in figures—in charts—and while a very interesting assumption, it leads no further than to saying that in each individual child there is an individual variation. Until this work is done carefully and in terms that can be expressed in figures we have not made a beginning.

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SOME CLINICAL FEATURES OF EPIDEMIC POLIOMYELITIS: PREVALENCE; TYPES; COMMUNICABILITY; MORTALITY.

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There is perhaps no disease whose literature has increased so rapidly in the last five years as acute poliomyelitis.

In a previous paper* published two years ago in conjunction with Dr. Frederic H. Bartlett, we endeavored to collect from medical literature all the reports of epidemics or outbreaks of this disease up to the year 1907. They numbered some 33, the total number of reported cases being 1,942, of which, however, 1,053, or considerably more than half the entire number, occurred in the Scandinavian epidemics of 1905 and 1906. The epidemics collected in this paper were mostly of small extent, the largest one, previous to 1905, being the Vermont epidemic with 132 cases. They were widely distributed both as to time and place, reports being drawn from almost every civilized country in the world. During the past three years reports have multiplied rapidly, indicating an alarming increase in the prevalence of this disease, especially in the United States. In the appended table, for the preparation of which I am indebted to Dr. Bartlett, we have endeavored to bring the reports of published epidemics down to the close of 1909. This record cannot be regarded as complete, for in many instances writers have referred to other epidemics of which no published report has yet been made.

The Scandinavian epidemics of 1905 and 1906 were followed by the great epidemic of New York City and vicinity in 1907, in which the total number of cases has been estimated at from 2,000 to 2,500.

The same year at Schenectady, N. Y., an epidemic with 29

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* *The American Journal of the Medical Sciences*, May, 1908.

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cases occurred. In Massachusetts an unusual prevalence was noted, with small outbreaks at Pittsfield (18 cases) and Haverhill (14 cases) and Boston (36 cases). While in western Pennsylvania four marked outbreaks were reported, at Oil City (50 cases), Du Bois (over 100 cases), Ridgway (30 cases), Eau Claire (14 cases), and other groups of smaller extent. Also an outbreak in Oceana County, Mich., with 20 cases.

During the year 1908 the disease again prevailed in western Massachusetts, especially Franklin County, where 69 cases were seen, with only 67 in the rest of the State. It also spread to the South and the West, particularly the Northwest. An outbreak in Salem, Va., was recorded with 29 cases. Several were seen in Wisconsin, the principal one being at Eau Claire, where there were 167 cases, with 185 in the rest of the State. In Minnesota an outbreak with 20 cases was reported at Northfield and another at Moose Lake and Barnum with 45 cases. In Iowa a small epidemic of 9 cases was observed at Whittemore, and the writer of this report states that "numerous other cases occurred throughout the State." From Michigan also reports came of several epidemics, the principal one being at Flint, with 30 cases. In the Northwest the greatest number of cases were reported from Minnesota: 83 cases in St. Paul, 45 in Carlton, 35 in Winona, 10 at Lewiston, 8 at Finlayson, and many smaller scattered groups. The disease assumed such prominence as to be recognized and made a subject of special investigation by several State Boards of Health, including Minnesota, Wisconsin and Massachusetts.

The year 1909 witnessed several small outbreaks near New York; one at Staten Island, one at Ironia, N. J., and quite a large one in a circumscribed district of the Borough of Brooklyn, in which about 150 cases were seen. In Nebraska there occurred at York and vicinity one of the most severe epidemics yet observed, with over 200 cases. Besides these larger outbreaks, reports of which have found their way into medical literature, many smaller ones are known to have occurred of which it is impossible to give any detailed information.

In addition to those which have been observed in our own country there was in 1908 an epidemic in Victoria, Australia, with 139 cases. In 1909 one occurred at Vorpommern, Germany, with 51 cases, and in Westphalia there were 436 cases occurring at twenty-one centers.

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Epidemics of Poliomyelitis, 1907-8-9.

Year and Season.	Place.	No. of Cases.	Deaths.	More than one in a Family.	Topography and Hygienic Surroundings.	Reference.	Remarks.
1907. July to October.	Vienna, Austria.	9	..			Zoppert. J. *Wiener Med. Woch.*, Vol. XLVII., 1908, p. 2563.	
September.	Eau Claire, Penn.	14	2	one instance of 3 in a family.		Urey, F. F. *Penn. Med. Jour.*, Vol. XII., 1908-09.	
Autumn.	Oil City, Penn.	50	..			*Ibid.*	
Summer.	Ridgeway, Penn., and vicinity.	50	4		Narrow river valley. Hygienic conditions poor.	Urey, *ibid.*, and Terriberry, J. F., *Long Island Med. Jour.*, Vol. I., No. 12, p. 490.	Season one of the dryest in history of state. 20 cases in radius of 50 miles from Ridgway.
Summer.	Dubois, Penn.	100	..			Urey, F. F. *Penn. Med. Jour.*, Vol. XII., 1908-09.	Pigs and chickens also affected.
Summer.	Lehigh Co., Penn.	9	..	2 instances of 3 in a family.		*Ibid.*	
July-October.	Massachusetts. Pittsfield. Boston. Haverhill.	18 36 14	.. .. ..	11 instances of more than 1 case in same family.	Hygiene excellent.	R. W. Lovett. *Boston Med. Surg. Jour.* Vol. CLIX., p. 131.	234 cases in entire state.
July-September	Oceana Co., Mich	20	..	2 instances 4 in same family. 1 instance 2 in same family.		W. L. Griffin. *Jour Mich. State Med. Soc.*, Vol. VII., 1908, p. 49.	
July-October.	New York City and vicinity.	2,000	..			*Jour. Nervous and Mental Diseases* Oct., 1909.	
Summer.	Schenectady, N. Y.	29	2			C. F. Clowe. *Albany Med. Jour*, Vol. XXIX., 1908.	
1908. April-June.	Victoria, Australia.	155	7		Hygiene fair. Very warm, dry summer.	H. D. Stephens. *Intercol. Med Jour. of Austria.* Vol. XIII, 1908, p. 573	Constipation a persistent symptom in most of cases. Epidemic not related to other diseases.
June-August.	Salem, Virginia.	25	3			R. M. Wiley. *Jour. Amer. Med. Assn.*, Vol. LII., p. 617, 1909.	Hot season and high humidity.
Summer.	Eau Claire, Wisconsin.	167	25	1 instance of 5 in family. 1 instance of 3 in family.		Manning, J. *Wisconsin Med. Jour.*, Vol. VII., 1909, p. 611.	29 cases traced to direct exposure. In the rest of the state 185 cases occurred same year.
June-November.	Franklin Co., Mass.	69	5	2 instances of 3 in family. 7 instances of 2 in family.	52 cases occurred in river valley. Hygiene: excellent, 4; good, 17; fair, 31; bad, 17.	Emerson, H. C. *Boston Med. and Surg. Jour.*, Vol. CLXI., 1909, p. 115.	24 cases in town of Colvain. 67 cases in rest of state same year. Practically all patients in good health at the time of attack.

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5.		Whittemore, Iowa.	9	..			Sams, J. H. *Iowa Health Bulletin*, Vol. XXII., 1908, No. 5.	Numerous other cases reported to have occurred throughout northern Iowa.
5.	July-September.	Northfield, Minnesota.	20	7	1 instance of 4 in family. 1 instance of 3 in family. 1 instance of 2 in family.		Strang, D. M. *Jour. of the Minn. State Med. Assn. and the N. W. Lancet*, Vol. XXIX., 1909, p. 297.	
7.	July-August.	Moose Lake & Barnum, Minnesota.	45	5	1 instance of 5 in family. 2 instances of 3 in family.		Hamilton, A. S. *Jour. of Minn. State Med. Assn. and the N. W. Lancet*, Vol. XXIX., 1909, p. 423.	Constipation and diarrhea equally common.
1.	July-October.	Flint, Michigan.	30	5	1 instance of 2 in family.		Manwaring, J. G. R. *Jour. Mich. State Med. Soc.*, Vol. VIII., No. 4, 1909, p. 161.	Total number of cases in this district probably 50.
1909.	Late Summer.	Vorpommern, Germany.	51	8	No instance.		Peiper, E. *Deutch Med. Woch.*, No. 9, May, 1910, p. 399.	No case could be connected with previous case. Many cases occurred in isolated houses in mountain districts.
1.	June-October.	Westphalia, Germany.	436	66			Krause, Paul. *Deutsch. Med. Woch.*, Vol. XV., p. 1812.	General statement made that some cases traced directly to exposure to affected persons and in others the disease apparently conveyed by third party.
1.	Summer.	St. Paul, Minnesota	83	7			Hill, H. W. *Jour. of Minn. Med. Assn.* and *N. W. Lancet*, Vol. XXIX., No. 17, p. 639.	No evidence of contagion in these cases. Persons attacked were especially healthy individuals.
1.	Summer.	Winona, Minnesota.	35	3			*Ibid.*	
1.	Summer.	Lewiston, Minnesota.	10	..			*Ibid.*	
1.	Summer.	Finlayson, Minnesota.	8	..			*Ibid.*	
1.	Summer.	Carleton, Minnesota	45	..			Frazer, H. G. *Jour. of Minn. State Med. Assn.* and *N. W. Lancet*, Vol. XXIX., 1909, p. 154.	
5.	July-September.	York, Nebraska, and vicinity.	Over 200	..	1 instance of 6 cases in 1 family; 3 instances of 4 in 1 family; 1 instance of 3 in 1 family; 2 instances of 2 in 1 family.	Hygiene was generally good.	Shidler, G. P. *Jour. Amer. Med. Assn.*, Vol. LIV., No. 4, p. 277.	Statistics for number of cases in family drawn from a detailed study of 60 cases. Most of those attacked in previously good health.
7.	Summer.	Brooklyn, N. Y.	150	..		Tenement district.	Kerr. *N. Y. State Jour. of Med.*, Dec , 1909.	Epidemic occurred in a district about one by one and a half miles in the city.

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Briefly to summarize the recent epidemics they were as follows:

1905 and 1906	Scandinavia, 2 epidemics, with total cases.	1,053
1907	New York City and vicinity, with total cases	2,000
1907	11 other epidemics, with total cases....	349
1908	8 epidemics, with total cases.....	520
1909	9 epidemics, with total cases....	1,018

making a total of thirty-one outbreaks or epidemics with 4,940 cases in the past five years, or more than five times as many cases as previously recorded in medical literature.

While it is certainly true that as a consequence of the greater interest in the subject, cases of poliomyelitis are now recognized which formerly might have been overlooked, still even a cursory survey of the facts above cited must convince one that a wave of infection, starting apparently from Scandinavia, is gradually spreading over this country, possibly over the world. It is hardly conceivable that such epidemics as some of those recently recorded have been previously passed over without recognition.

Types.—The next question to which I wish to ask your attention is: Has the disease known as poliomyelitis changed in type, and is there an essential difference between the sporadic and epidemic form of the disease? Without going fully into the arguments *pro* and *con,* I wish to state my belief that there is no essential difference between the two forms of the disease except possibly in communicability and severity. The analogy to cerebrospinal meningitis is a very close one. No one I think now denies that sporadic and epidemic cerebrospinal meningitis are the same disease. This is established by both bacteriology and pathology They differ in that the mortality of the sporadic form is lower and communicability apparently much less; in fact, it is in most instances impossible to establish any connection between cases. It seems probable that both of the differences referred to may be explained by a difference in the virulence of the infection in the sporadic and the epidemic disease.

All this, so well known regarding cerebrospinal meningitis, applies I think quite as well to acute poliomyelitis. Evidence of contagion in sporadic cases has, it is true, very seldom been found; but only recently has it been even suspected. Fatal cases

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of the sporadic form are however not very rare, but often, I think, wrongly diagnosed. Epidemics of poliomyelitis thus far reported have invariably occurred in the summer months, while sporadic cases, though far more frequent in the summer, are observed throughout the year. A practical demonstration of the identity of sporadic and epidemic poliomyelitis would seem to be afforded by the experiment of Dr. Flexner, who had no difficulty in transferring to a monkey the disease from the organs of such a sporadic winter case. We must I think admit that there is no essential difference between the two types of the disease, since they have apparently the same cause, the same symptoms and the same lesions.

It would appear quite evident from published statistics that the virulence of the infective agent of poliomyelitis is greater when the disease is epidemic, and that this may explain both the higher mortality and the more ready communicability then observed; in other respects the epidemic and the sporadic disease seem the same. Not only is a difference in severity seen between the sporadic and the epidemic forms, but the disease varies much in severity in different epidemics, and in the same epidemic at different times. Thus it was noted in the Nebraska epidemic that the cases were more severe and the mortality higher in the latter part of the epidemic than in the beginning. A similar variation has also been observed in epidemics of cerebrospinal meningitis.

Mortality.—It is difficult to make any statement regarding the mortality when the disease occurs sporadically. A suggestion may be obtained from the Massachusetts reports for 1907. Of 234 scattered cases reported throughout the entire state, the mortality was 5 per cent. There were included only two outbreaks of any considerable size—one of 18 and one of 14 cases. In our paper published two years ago, of 1,659 cases in epidemics, the mortality was 12 per cent. Of 14 more recent, localized epidemics which have been pretty fully reported, representing 1,209 cases, the mortality was 12.3 per cent. This correspondence is certainly very striking. The inclusion of abortive cases will modify mortality figures. In the recent epidemics their occurrence has generally been recognized, and they are usually included in the statistics. Some observers, believing strongly in the abortive form, report many such cases, others, more skeptical, include but very few. As an example of the former class Brorström may be mentioned, who reports an epidemic in Sweden in 1905-1906, with

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394 cases, of which only 79, or not quite 20 per cent., had paralysis; while H. W. Hill, writing up the Minnesota epidemics for the State Board of Health, excludes all cases but those with paralysis. With this uncertainty regarding diagnosis it is at present impossible to state what the exact death rate is. In various epidemics it has ranged from 6 to 20 per cent., the average being about 12 per cent.; in sporadic cases the mortality is certainly much lower.

Abortive Cases.—The belief in the occurrence of abortive cases is of recent origin. Up to five years ago the idea would have been scouted. Cases without paralysis would unquestionably have been excluded from the classification; while cases with paralysis ending in recovery would have been, for the most part, classed as at least extremely doubtful. Clinical evidence, however, has so rapidly accumulated upon this point during the past few years that we must admit the occurrence of both these types.

The existence of cases in which complete recovery takes place even after paralysis develops has been well recognized during recent years. In the New York epidemic of 1907 numerous instances were observed and reported. This has been confirmed in the epidemics of Scandinavia and in many other places.

Cases in which there is no paralysis, or abortive cases, must be subjected to closer scrutiny. Occurring apart from an epidemic, or in a family without other cases, they would on clinical grounds properly not be accepted. But when in families abortive cases are preceded by, or are followed by, paralytic cases, and when this happens repeatedly and regularly, this type must be taken into account. Now that the infectious nature of poliomyelitis has been established, analogy would lead us to expect the abortive types just as in cerebrospinal meningitis and in other infectious diseases. There seems no doubt, from clinical observations, that they actually occur. Numerous examples occurred in the Nebraskan epidemic and in that of Tröstena, where two or more children in a family had typical attacks and others with a similar onset and early symptoms recovered promptly and completely in the course of a few days without any paralysis, while in some others only a brief muscular weakness was noted. One thing must be strongly emphasized, that at present on clinical evidence we can include as abortive types of this disease only those closely associated with the paralytic cases. It is highly probable,

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however, that in epidemics they do occur quite apart from such association.

The symptoms of the abortive cases, while fairly uniform, are not sufficiently characteristic to enable the diagnosis to be made of these cases when seen by themselves. The onset is usually abrupt, often with vomiting; there is constipation and fever, usually from 101° to 103° F. Accompanying these general symptoms there is very often hyperesthesia, pains in the back and neck and in many some stiffness of the neck is observed. There is general prostration, which may be sufficient to keep the patient in bed. The duration of these symptoms is usually only two or three days, after which they rapidly subside and the patients completely recover.

Occurring sporadically such cases would not be diagnosticated. They would hardly be suspected. In epidemics they would be recognized only by their association with paralytic cases. The proportion of abortive cases cannot now be stated, or even estimated. Undoubtedly a much larger number occur in epidemics than the records indicate, for most writers in their statistics have been inclined to exclude doubtful cases, in which group certainly many of these would fall. It seems probable, however, that the number is quite large. Wickman records 23 such in the Tröstena epidemic of 49 cases. In the Nebraska epidemic of 66 cases studied, Shidler states that 10 had no paralysis and 11 had only incoördination. Many of the milder cases occurring during an epidemic would not even be seen by physicians. It is enough at present for us to recognize their existence and, I believe, their frequency. If Dr. Flexner's observations upon the cerebrospinal fluid in the early stages of the experimental disease in monkeys is confirmed by observations upon man, the status of these abortive cases will be definitely settled.

Communicability.—In our previous paper the ground was taken by us that existing evidence strongly suggested, but did not demonstrate, that the disease was contagious. Since that time several important contributions have been made to this part of the subject, notably the reports of the epidemic in Tröstena, Sweden, by Wickman and that of the York, Nebraska, epidemic by Shidler.

The opportunity for a study of the spread of the disease in Tröstena was very unusual. The parish of Tröstena embraces a district of about twelve square miles. It is off the main lines of travel and communication with the outside world is very limited.

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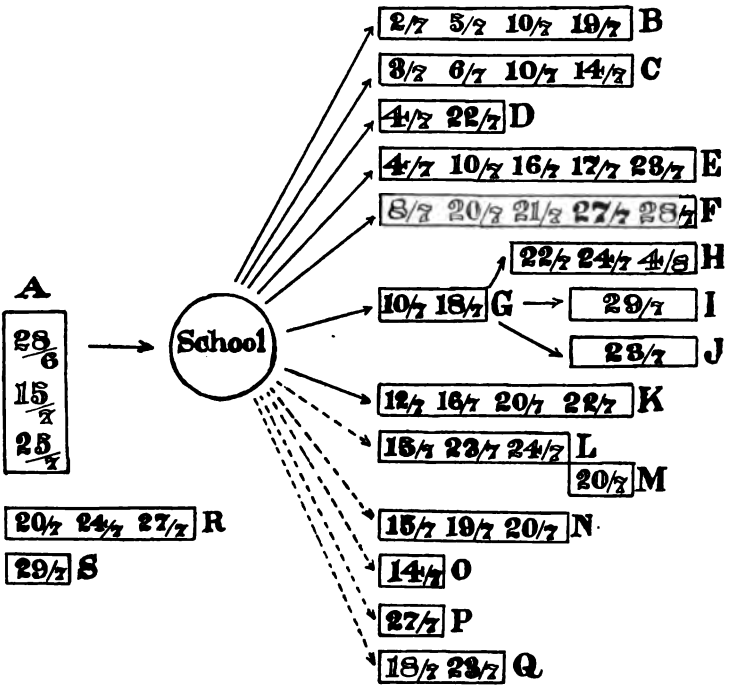
The entire population was about 500 persons, living in detached houses, which for the most part were quite isolated from each other and with very little intercommunication. In this community there occurred within a period of six weeks 49 cases, affecting thus nearly 10 per cent. of the population. The water supply of the families was from individual wells and the milk supply from separate cows owned by the different families. The food for each



TRÖSTENA EPIDEMIC OF POLIOMYELITIS AFTER WICKMAN.
The figures give the month and day of the development of the different cases. The paralytic cases are indicated by heavy type and the abortive cases by lighter type. The lines indicate the association of the families with the school.

household was largely from the products of each separate farm. The spread of the infection seemed clearly traceable to the parish school.. The relation of the families in which the disease occurred to the school and to each other is shown in the accompanying chart, which I have slightly altered from Wickman.

The first case occurred in family A. This child was taken ill June 28th, but attended school that day. The disease developed in seven families respectively on July 2d, 3d, 4th (in two families),

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8th, 10th and 12th, affecting in every instance children who had been at school with the sick child in family A. They were also the first children in each of the seven families (B., C., D., E., G., F., K.) to be attacked. The only families in the parish in which association with the school could not be traced were R. and S., in one of which three persons were attacked. In families L., N., O., P. and Q. there were children who attended the school but who did not themselves develop the disease. They were apparently the carriers of the infection to their homes. Families H., I. and J. were apparently infected from G., while M. lived very near to L., and free communication existed between the households.

Another instance of the probable transmission of the disease through a third party occurred in the neighborhood of Tröstena. A child of three years with his mother visited at a home near where was also visiting a servant from another house in which 2 cases of poliomyelitis had occurred. Four days after arrival the boy came down with the disease. He was taken to his home, one day's journey distant, and from him a new group of cases developed; the boy's father in nine days, and another child in the same house in sixteen days after his return.

The Nebraska epidemic, though not so fully reported, affords many examples of direct contagion. Very striking, as in the Swedish epidemic, were here the instances of a number of cases in the same family. Thus there were 6 families with 2 children attacked, these being the only children in the family in 3 of these; 5 families with 3 cases each, in every instance all the children of the family; 3 families with 4 cases each, these being all the children in 1 of the families; and 1 family in which every one of 6 children was attacked.

Some special groups of cases in this epidemic also afford rather striking evidence of contagion. Thus the first child in York to be attacked was a boy of five, who came down with poliomyelitis just nine days after the return of his mother and infant brother from a visit to a house in another town where was a child suffering from the disease. This seems a pretty clear case of transmission by a third party.

Next door to this first case in York lived a family with 6 children, all of whom, one after another, came down with the disease. To this household with 6 cases came a child who mingled closely with the affected children; five days later he was attacked and died in three days with bulbar symptoms.

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Another series of connected cases indicates conveyance of the disease from a patient some time after the acute symptoms had passed off or possibly infection from premises. A child entered the infected district, contracted the disease and was taken to his home in the country; subsequently a twelve-year-old sister came down with the disease. "Several weeks later" an infant was brought from another town where no cases had occurred and spent the night in this house; five days after, this child was attacked. "One months later" another child came from the country to the affected house, spent a single night, and five days later developed fever, followed in three days by paralysis of both legs.

The above examples have all been drawn from cases occurring in children. Shidler narrates one group of cases in which transmission among adults seems to have occurred in the same way. A member of a threshing crew of 7 young men was exposed to the disease in a household where there were 2 cases and had a slight attack. One after another the entire crew developed the disease, which in 1 case ended fatally. The men were thrown very closely together, ate and slept together, drank from the same jug, etc.

Illustrations affording evidence of the spread of this disease by contagion might be greatly multiplied did space permit. It seems to me to be conclusively shown that this disease under certain conditions is highly contagious, demanding during an epidemic strict quarantine. It would seem, also established by evidence, almost, if not quite, as conclusive that healthy persons may be carriers of infection, and that this may take place for a considerable time after contact with active cases. No evidence whatever is afforded by clinical reports of any medium of contagion other than contact with persons suffering from the disease or those exposed to it.

It has been my purpose in the above paragraph to emphasize the facts recently brought out which bear on the question of contagion, citing those chiefly which support this view. It should, however, not be forgotten that in a very large number of the epidemics reported, it has been impossible to trace a connection of one case with another, though careful search for such connection has been made. Thus Piper, in the Vorpommern epidemic of 1908, states that in this epidemic of 51 cases, with a mortality of 17 per cent.—this indicating its severity—there was no instance in which a case could be traced to a previous one, and no instance

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of more than 1 case in a family. H. W. Hill, in his report upon the Minnesota epidemic, mentions 61 primary cases and only 12 secondary cases in families in a total of 81 investigated. Thus the disease apparently varies greatly in its communicability at different times. These differences at present we are unable to explain or to reconcile.

A consideration of the recent knowledge, pathological, clinical and experimental, which has accumulated regarding this disease, makes it evident that a new name must be sought. About twenty-five years ago the common designation of "acute infantile paralysis," or "infantile spinal paralysis," was superseded by one based upon the anatomic lesion, as then understood, and since that time the disease has been most frequently called in medical literature "acute anterior poliomyelitis," which has been shortened into "acute poliomyelitis." We now know that this term is not exact, and, in fact, not descriptive, for the disease is not limited to the anterior horns, nor to the gray matter, nor even to the cord. To speak of the cerebral or bulbar form of poliomyelitis, or any myelitis, seems rather absurd. Following analogy in epidemic cerebrospinal meningitis, I venture to suggest the term "epidemic myelitis," or, better, "epidemic myeloencephalitis," as one which is fairly descriptive and preferable to the various personal names which have recently been attached to the disease.

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EXPERIMENTAL EPIDEMIC POLIOMYELITIS AND ITS RELATION TO HUMAN BEINGS.

BY SIMON FLEXNER, M.D.,
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In addressing the two societies on the subject of "Experimental Epidemic Poliomyelitis," I shall confine myself to the essential points of this investigation, with especial reference to the clinical data that have been so well presented by the two speakers preceding me. As you now know, in the last few months it has been demonstrated with certainty that a disease agreeing both in clinical types and in pathologic symptoms with anterior poliomyelitis can be produced regularly in certain of the lower animals; and my own opinion is that the knowledge of this disease has been greatly extended by the fact of this transmission, and that the hope of the final conquest of the disease lies, perhaps, along the direction of this experimental work. I trust that you will not consider that I overemphasize this point, because I am myself only an experimentalist; but in view of the history of the disease, it seems that the hopeful aspect comes from this circumstance (as has been the case with other diseases that were formerly entirely baffling): that it can be transmitted regularly to the lower animals, in which it can be studied in all its relations.

This transmission, up to the present time, has been accomplished only in connection with the higher of the lower animals; that is, in the monkeys, including the anthropoid apes and the lower species. It is a perfectly simple thing to do. It is merely necessary to bring some of the central nervous system (perhaps, best, the spinal cord) of an infected human being who has succumbed to the disease before it has become chronic into intimate relationship with the central nervous system of these animals. In our own experiments we have employed the spinal cord from 3 children, who succumbed in the first week or two of the disease; and have made the inoculations directly into the brain, with some few exceptions, making a small trephine opening into the skull, and injecting minute quantities of a suspension of this matter directly into the substance of the brain. The animals having been anesthetized, there was no discomfort.

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There are no immediate effects following the injections. The animals quickly recover after the effects of the anesthetic disappear; and for a variable period they are perfectly well. Then, after the expiration of this period of time, which is calculated as the incubation period of the disease, they almost without exception come down with symptoms comparable in all respects with those of the human disease. For this statement we have the evidence of those who are familiar with the human disease and have been kind enough to study these animals, especially Dr. Holt, who has given considerable time to it, and who states that the clinical appearances are indistinguishable from those of the human disease.

Although this is the most certain method of transmitting the virus, it is possible to transmit it in other ways. Indeed, there are no ways, I think, in which the virus, having been introduced beneath the skin or into the mucous membranes, may not set up the disease. It can be produced by injecting into the peritoneal cavity, into the subcutaneous tissue, or directly into the circulation; but these means are less certain than is the means of direct inoculation into the nervous tissue. By means of the latter method the disease can be produced in from 90 to 100 per cent. of all animals infected. By the other modes of infection the disease develops very irregularly; and it has not been possible to continue throughout a long series the transmission of the virus by any other method than that of direct inoculation into the nervous system, with one exception, to which I will refer in a few moments.

The spinal cord and the brain of animals succumbing to the disease, or those of animals killed during the first week of the disease or after a somewhat longer period, are just as infectious as was the original human virus. The question, therefore, arises: In what does this infection consist? We have made a most painstaking study of the materials, the nervous system and other parts of the body, by every means which we can now command, for the purpose of demonstrating a visible microörganism, and have not succeeded in finding anything that would account for the symptoms and conditions produced. That the active agent is a living virus must be assumed from the fact that this transmission can be continued over a long, and perhaps an indefinite, period of time, and through an indefinite number of animals. We possess viruses that have been transmitted through twenty generations of monkeys and are now more active than at the beginning. It is not conceivable, therefore, that this matter that we originally used

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contained a soluble injurious agent, which has gone on reappearing in the successive series of animals, and which we are still transmitting in a sufficient state of concentration to produce these results. We believe that the virus is a living thing, transmitted as a living organism from one animal to another, and in sufficient amount in each case to produce the characteristic effects of the disease.

It became apparent, in the course of the efforts made to discover the nature of the virus, that it is extremely minute. If suspensions of the nervous system containing it are passed through the closest filter, the clear filtrate is found to be quite as active as the original emulsion—how active, may be illustrated by the fact that if a quantity equivalent to one-hundredth of a cubic centimeter of this fluid, which is a mere extract of the nervous system, is brought into the brain of a monkey in the manner described it will produce the disease. Therefore, I may say that the indications are that this active agent belongs to the class of so-called filterable viruses; that is to say, viruses of ultramicroscopic size, so small that they cannot be revealed by the most powerful microscope. They cannot be discovered by means of the ultramicroscope, which shows minute particles of proteid in solution or in dilute serum. Moreover, even if these poisonous substances can be seen in these fluids under the ultramicroscope, there is no way of distinguishing them from the protein molecules therein contained.

We believe that the virus is a filtrate virus, of which we know other examples, causing destructive diseases. At one stage of yellow fever in human beings the virus is filterable; and the virus of dengue is also filterable. The virus of rabies is filterable, but it must be relatively much larger than the virus of poliomyelitis, because it filters with difficulty, and only through relatively coarse filters; whereas that of poliomyelitis is filtered with ease through the finest filter of which we have any knowledge.

We have no complete evidence of its being possible to cultivate the virus outside the body. We have obtained changes in culture-fluids, indicating increase of the virus; but we have not been able to transmit the disease by means of such fluids. The reason for this is not clear. One possibility is that we have not really cultivated the virus; but that the changes observed were produced by transferred ferments, and another is that the virus does not stand artificial conditions of growth very well, but is adapted

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only for a parasitic mode of existence. When forced to undergo the saprophytic one, therefore, it loses its pathogenic properties. This, however, is mere speculation. We do not really know that the cultivation has been actually accomplished.

I mentioned, a short time ago, that the disease as produced in monkeys presents the clinical aspects of the human disease. It should be added that it is a highly fatal disease in these animals. There is no real comparison between the fatality of poliomyelitis in monkeys and in human beings. You have learned how variable is its mortality in human beings during spontaneous epidemics. While it has been reported to be as great as 30 per cent., or even more, the average mortality is nearer 5 per cent. In monkeys, on the other hand, it is from 50 to 100 per cent. In the first 80 monkeys infected, the mortality was 50 per cent., but recently we have not been successful in saving a single monkey that developed the disease—the mortality, therefore, being 100 per cent. In other words—I place emphasis on this point—the experimental disease is not only identical with the spontaneous with respect to its clinical appearances, and also in respect to its pathologic lesions (since the study of the central nervous system microscopically has developed the fact that the lesions of the disease in the monkeys are not to be distinguished from those in human beings), but also that the effects of the experimental disease are much more intense upon these animals than those of the spontaneous disease are upon human beings.

Now the varieties of the spontaneous disease are reproduced in the experimental disease. The distribution of the paralysis in the experimental disease is the same as in the spontaneous; the occasional involvement of the brain, the involvement also of the higher centers, all occur in the experimental disease, and perhaps with about the same frequency as in the spontaneous.

The question of the incubation period in animals has been given attention, because it was believed that it might throw some light upon the incubation period in human beings, as well as upon the so-called sporadic cases in human beings. In monkeys, by the direct mode of inoculation, the period varied from three to thirty days, the average period being seven, eight, or nine. The animals sometimes develop the disease about three days after inoculation. Of far greater interest, however, is the fact that the animal inoculated with the virus may remain in good health until thirty or more days after inoculation, when the symptoms suddenly appear

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unmistakably, and the pathologic examination shows the animal to have acquired the disease; further inoculation showing that the virus survived in living form. What we speak of as the sporadic cases of the disease in the intervals between epidemics, but not occurring altogether independently of epidemics (the latter point not having been sufficiently studied), may, after all, not be cases of reinfection; for instance, the cases appearing now, or two or three months ago, in localities in which there were a number of cases during the last summer, may have been cases in which the infection occurred at the time of the epidemic, but which, for some reason not yet understood, have been delayed in respect to development until circumstances favored the breaking out into unmistakable examples of the disease.

Let me again draw your attention to rabies, because there are similarities in the lesions in these two diseases. Recall how long a period may elapse between the introduction of the virus and the development of the symptoms of rabies. I should not, therefore, be so sure that these examples of the sporadic occurrence of poliomyelitis do not represent, similarly, cases of infection and of delayed appearance of the characteristic symptoms.

I have mentioned the fact that there is one other mode of infection of this disease that gives results almost as constant as those following the method of direct inoculation into the brain. The number of instances observed is not yet so great as with the direct cerebral inoculation, but the results are much more constant than those following any other mode, except the intracerebral, by which the virus is brought into the body. We have been greatly interested in the question as to how spontaneous infection in human beings takes place, and have recently concluded that, in all probability, the virus being of the nature that I have described, it does not lead a prolonged saprophytic existence outside the body. Therefore, we have assumed that it is kept alive in the body, and that its chief place of increase is the central nervous system; because that is where it develops and produces its effects, the remainder of the body being only slightly involved in the pathologic process. For this reason we have considered the channels through which the virus established in the central nervous system leaves the body.

There are several possibilities. The virus might leave with the ordinary excretions, from the kidneys, intestines, etc., but we have not found it in the urine or the dejecta. There would be no difficulty in finding it there if it were present. Since it is

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filterable, all bacteria can be excluded. We have also taken into account the direct and intimate connection existing between the membranes of the brain and the nasal and pharyngeal mucosa, and have thought it possible that the excretion of the virus might be through these channels. We have removed the mucosa of the nose and throat, including the tonsils from animals succumbing to the disease, breaking it up into particles and suspending this emulsion; and have found the filtrate to be active. The mucous membrane of the nose and throat, therefore, contains the virus. Its containing it might, however, be merely accidental, in connection with the blood that these parts contain; but we have satisfied ourselves that the blood is not highly infectious in the height of the disease. It is possible to produce the disease in other animals by injecting a large amount of the blood, but small amounts do not set up the disease. We believe, therefore, that the blood can be excluded as a carrier of the infection. In all probability the membrane of the nose and throat becomes infected more severely because of the close proximity to the membranes of the brain and cord than other parts of the body, and because of their direct connection. In other words, the poison probably leaves the body in this situation in active form. If this view is true in the human disease, it points at once to the application of practical preventive measures that will destroy the virus in those infected and prevent its being communicated to other persons.

So much having been gained, the question arose whether the virus could be made to enter the body in the opposite direction. Having in mind the way in which infection takes place in epidemics, and having established by means of pathologic studies that the first lesions occur in the membranes of the brain and cord, we found that the membranes become infiltrated with cells of the mononuclear type, and not by the polynuclear leukocyte. We also discovered that the paralyses are really accidental effects of the disease; that the poison does not exert its action against the ganglion cells, but simply interferes with the circulation; and that it is because of the interference with the circulation that the paralysis occurs. It seems that the membranes are the seat of the primary lesion. The close lymphatic connection existing between the nose and the membranes of the brain might be the path through which the virus finds entrance into the brain, and this idea was put to the test.

The intact nasal mucosa in the monkey has, thus far, not been

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found subject to inoculation that leads to the production of the disease, but it requires merely a small amount of traumatism to open up the way and lead to infection. Other membranes do not behave in this way; there is something peculiar in this membrane that renders it highly susceptible. The virus travels from the nose to the meninges along the olfactory nerves, and gains entrance to the membranes of the cord and localizes in the manner so familiar. I have a dissection of an animal infected in this way, showing the lesion. The nose has not been opened, but you can look in from above and see the hemorrhagic lesion and the close proximity of the olfactory bulbs to the nasal mucosa. The connection is bridged by the olfactory nerves, and the separation is only a line. It is a direct mode of connection between the two structures. You will see the congestion developing about the meninges. This animal came down with the disease three days after the inoculation of the nasal mucosa.

These are points of such practical significance that I trust I have not overemphasized them. I intended to emphasize them, because you are the ones who have to deal with the disease in man, and perhaps these facts developed through experimentation may be utilized by you. There are also one or two other points of interest.

Dr. Holt alluded to the fact that there are changes in the cerebrospinal fluid of the monkey. These changes are unmistakable. Before the appearance of the symptoms you can tell by this means which animals will come down with the disease. Often, a number of days before the symptoms appear, the cerebrospinal fluid is altered. The protein increases, and this fact can be demonstrated by any method that shows increase in protein. There is also an increase in cells—partly polymorphonuclear leukocytes, and partly mononuclear leukocytes; but you do not have a turbid fluid that arouses your attention, but a clear fluid. Specimens have been taken from the cerebrospinal fluid in children, and found clear and to contain an excess of lymphocytes; but these observations were made late in the disease. If, however, lumbar puncture is made day by day, there is a point in the disease in monkeys at which the fluid is changed perceptibly in ocular appearance. This period in human beings is, of course, overlooked, because it lasts only one or two days, and then the fluid returns to its previous condition. The paralysis, however, has then al-

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ready set in. This period coincides with the interval just before the paralysis, and does not last long after the paralysis is established—which I take to mean that the height of the disease is reached at the period of the paralysis, after which there is recession of the pathologic process, the damage having already been done.

Now there is another point of practical interest, namely, that the animals that have recovered from the disease are not subject to inoculation. An attack of the disease appears to afford immunity. It would be interesting to learn whether the same is true of the disease in human beings. This is not the only point of great significance with reference to this fact. It has been found that the blood of the animals recovering and having immunity is capable of neutralizing the virus. When the virus is mixed with the serum and injected, the animals do not develop the disease. We have also been able to show that the blood of children who have recovered from poliomyelitis also possesses this power. To one with animals at his command this discovery leads to another form of experimentation. If it is possible to neutralize the virus by direct mixture outside the body before inoculation, the question at once arises, Is it possible to neutralize the virus after inoculation into the nervous system? It is possible, but to what extent I cannot say, because our experiments have not been sufficient in number or in duration to allow of my making a statement. I may say, however, that in animals and persons recovered from the disease there remains for a long time in the circulating blood a substance neutralizing and antagonizing this virus. This is a hopeful situation. Should means be found to make an early diagnosis of this disease in human beings, as in monkeys, some influence may be exerted over the ultimate development of it, now that we have in our hands a definite antidotal substance. Much will depend upon our ability to increase the value of this antidote by subsequent immunization, and upon our ability to find lower animals capable of immunization. It is too early to say whether anything can be accomplished in this direction or not, but what has been stated is none the less of great significance. It is probable that only during the prevalence of an epidemic would one's attention be sufficiently directed to the disease to make the diagnosis in advance of the paralysis; but from what has been said about the cases that are abortive or entirely recover, and in view of the fact that the

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meninges and the vessels are chiefly affected, it is not outside the bounds of possible human achievement to influence the disease favorably under the circumstances mentioned. Taking into account what I have said, that the subject is in a very early stage of development, I may still add that I think the disease will come to be influenced favorably by the use of some antidotal substance. Since in monkeys the disease is so much more severe than in human beings, if we can interrupt the process in them there is greater hope that it can be interrupted in human beings.

The situation in this case is reversed. It is usually easier to affect favorably an animal disease, which is experimental, than a human disease, which is natural to the species. The conditions are reversed, since the experimental disease in this instance is so much more severe than the spontaneous disease. We have observed no example of the spontaneous communication of the disease to a healthy monkey. In view of what has been said about the communicability of this disease in human beings, with which I wholly concur, it seems strange that there is no evidence of that kind in respect to animals. Perhaps the reason is that it is impossible to make a direct infection by means of any channel, unless some injury is instituted, as, for example, the scarification of the nasal mucosa, or the penetration of the dura and the brain substance in inoculating the brain. Therefore, these animals are not naturally subject to this disease. We have to create a traumatism in order to produce it. That point may suffice to explain why this communication has not taken place, although under our experimental conditions the opportunity for direct transfer of the virus was frequently present.

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PARALYSIS OF THE NECK AND DIAPHRAGM IN POLIOMYELITIS.*

BY IRVING M. SNOW, M.D.,

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The knowledge of the nature and symptoms of poliomyelitis has expanded enormously within the last five years. New ideas of the communicability and pathology of the disease have been accepted. Landry's paralysis, polioencephalitis, and various painful paralyses are now included in one disease-concept. It is therefore with the hope that an unusual symptom-complex of poliomyelitis may find a place in the literature of the disease that I report a case of high cervical poliomyelitis causing a paralysis of the diaphragm and neck muscles and terminating in complete recovery.

The existing literature on this subject is very scanty.

Neck paralysis in combination with loss of power in the extremities is not unusual; on the contrary, isolated neck palsies are excessively rare in poliomyelitis.

Paralysis of the diaphragm is scarcely mentioned in treatises on poliomyelitis except with the fatal type of Landry's paralysis.

There is but one recorded case (Erb's†) of a combined paralysis of the phrenic and spinal accessory nerves existing as the sole lesions of a high cervical poliomyelitis. The origins of the spinal accessory and phrenic nerves are close together on the upper cervical cord. The spinal branch of the spinal accessory originates in the anterior horn of the cord, as low as the sixth cervical nerve. It is simply a motor spinal nerve, which has the unusual course of ascending into the skull and of leaving it again with a cranial nerve. In the neck the spinal accessory inosculates with the second, third and fourth cervical nerves and forms a plexus which innervates the sternomastoid and the trapezius muscles.

The phrenic nerve—fourth cervical nerve—arises from about

* Owing to lack of space, this article is here abbreviated by the omission of citations from the literature of the subject. The complete article may be obtained from the author.

† Erb: *Deutsch. med. Wchnschr.*, 1906, p. 1,899.

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the same tissue and innervates the diaphragm. Thus the two pairs of nerves originate in about the same area of spinal gray matter, and a focus of inflammation in the cervical cord might easily injure the functions of both phrenic and spinal accessory nerves.

The rarity of paralysis of the phrenic nerve may be due to the resisting power of its tissues to disease.

With paralysis of the neck muscles the symptoms are characteristic. The patient cannot rotate the head, hold it erect or raise it from the pillow. In sitting or standing, the head falls forward, backward and sidewise, not following the movements of the body; the head wabbles around on the body like the headpiece of a disabled marionette.

In Wickman's analysis* of the distribution of paralysis in the Swedish epidemic of poliomyelitis in 1905 with 479 cases, neck paralysis existed in 9 patients. This does not include cases described as Landry's paralysis or paralysis of respiration, or cases of ascending paralysis, in which all four extremities and the neck were paralyzed before the fatal issue. Thus, palsy of the neck muscles occurred in 1.9 per cent. of cases of the benign type of the disease; in only 1 case was there an isolated palsy of the neck. With the others loss of power in the neck was associated with paralysis of the extremities.

From the report† of the Collective Investigation Committee of the New York epidemic of poliomyelitis of 1907, one would conclude that loss of power in the neck is common. In 679 cases loss of motion in the neck occurred as the first symptoms in two instances, and at the height of the disease 299 patients (39.8 per cent.) could not hold up their heads. As to this symptom, both the questions of the committee and the resulting answers are vague as to whether the inability of the patient to raise his head came from general prostration or from paresis of the sternomastoid and trapezius; yet a fair conclusion is that in a considerable number of cases there existed a motor palsy of the neck.

It is interesting to note that the committee made an analysis of the residual disability in 318 cases in the same epidemic of poliomyelitis and no mention is made of a neck palsy, so that one may draw the conclusion that this symptom generally disappears. That with the neck paralysis of poliomyelitis a complete

* Wickman: *Heine-Medinschen Krankheit*, pp. 139-276.

† Report of the Collective Investigation Committee on the New York Epidemic, 1907, p. 45.

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recovery generally follows may also be inferred from the last edition of Starr.*

In an analysis of 1,292 permanent palsies from poliomyelitis collected from the statistics of Starr, Duchenne, Seligmuller, Sinkler, Lovett, no paralysis of the neck is recorded. Starr, however, states that during the disease the neck muscles are occasionally weak and that the paralysis may extend to the neck, throat, face and even the eyes, giving rise to the symptoms of bulbar palsy, the polioencephalitis inferior and superior of Wernicke.

Oppenheim and Zappert† do not mention paralysis of the neck or diaphragm in their article on poliomyelitis.

La Fétra‡ states that in 63 cases rigidity of the neck existed in 11 cases and a paralysis of the neck and larynx in 1 case.

Buzzard§ says that in poliomyelitis there is occasionally a rapid atrophic paralysis of the muscles of the head and trunk; and Holmes states that paralysis of the diaphragm is, in the majority of cases, only part of a general peripheral neuritis or poliomyelitis.

PARALYSIS OF THE DIAPHRAGM—PHRENIC NERVE.

Wickman¶ says that the pathologic disturbances of the mechanism of respiration in poliomyelitis are partly due to an affection of the spinal center of the intercostal muscles and diaphragm, and partly to a lesion of the origin of the vagus nerve. It is not always possible to distinguish whether the respiratory distress comes from a high cervical or a pontine source. An injury to the pneumogastric of medullary origin usually shows itself by recurring attacks of dyspnea and tachycardia.

With an isolated bilateral paralysis of the diaphragm the symtoms are usually very clear. The respiratory abdominal movements are the reverse of normal. In health during inspiration the abdominal viscera are depressed and the abdomen is protruded. With a diaphragmatic palsy the upper abdomen recedes during inspiration and is pushed out during expiration. Breathing is labored and difficult. Any effort causes cyanosis and dyspnea. The lungs are nevertheless clear of pathologic sounds.

* Starr, M. Allen: "Diseases of the Nervous System," 1908, p. 270.
† Pfaundler and Schlossman: "Diseases of Children," IV., p. 172.
‡ La Fétra: Transactions American Pediatric Society, 1908, p. 164
§ In Osler's "Modern Medicine," VII., 258.
¶ Wickman: *Heine-Medinschen Krankheit*, p. 34.

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The prognosis is always serious, partly from the danger of pulmonary infection, and is especially grave if associated with bulbar symptoms. If the paralysis of the diaphragm is complicated with a loss of power of the intercostal muscles the patient will probably suffocate. This danger is greatest in the first week, after which time the outlook improves.

REPORT OF CASE.

The history of my case of cervical poliomyelitis causing paralysis of the neck and diaphragm is as follows:—

Patient.—John D. was a boy of four, a patient of Dr. Villiaume. Previous to his illness a baby brother, breast-fed, had sickened August 29 with fever, vomiting, diarrhea and general tenderness, and died in a few days; and a cousin, also in the same house, was attacked with symptoms of meningitis and taken to Ohio. Both these were probably cases of poliomyelitis spreading by direct contagion. John D. sickened September 4th, with pain and tenderness in the limbs, trunk and neck, vomiting, fever, 104°F., pulse 150, and retention of urine. Later the pain and tenderness lessened, but there was still soreness and muscular twitching in the limbs, and in a day or two a flaccid palsy developed in the muscles of the neck. There were no convulsions; the boy was slightly delirious at times.

Examination.—I saw the patient first September 8th, the fifth day of the disease. He was a well-developed boy, lying in a reclining chair, breathing with great difficulty. The pulse was 110, regular; respiration 42. The lungs were clear; there was a complete paralysis of the neck muscles, so that the patient had no control of his head. The head fell backward and forward and sidewise unless held with the hands or resting on a pillow. The neck was not tender. Its muscles did not respond to the galvanic or faradic current. Evidently there was present a paralysis of both spinal accessory nerves injuring the function of the sternomastoid and trapezius muscles. Most of the child's distress arose from difficulty in breathing. The whole chest moved with great effort during respiration. With inspiration there was slight recession of the anterior ribs and the upper abdomen was drawn in; it bulged out at expiration, indicating a bilateral paralysis of the diaphragm. The right knee-jerk was absent, the left normal; sensation was normal.

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Course of Disease.—In the evening there developed great cardiac depression, rapid pulse and intense dyspnea, giving rise to the fear that the myelitis had invaded the medullary tract. This distress passed away in a few hours. After this for ten days the child remained in stationary condition and was fairly comfortable, except that the respiration was labored and rapid and that the neck muscles were useless.

September 17th: Fourteenth day of illness. Dr. James W. Putnam in consultation. There was no fever; respirations 40, pulse 120 to 140. The face was often flushed; the pupils were evenly dilated; the lungs were clear; the heart sounds good. The neck muscles were still paralyzed; did not respond to faradism. The abdominal muscles responded to faradic current; the upper abdomen receded at inspiration. Evidently the condition was due to a high cervical poliomyelitis affecting a focus at the origin of both pairs of phrenic and spinal accessory nerves.

October 21st: Forty-seventh day of illness. There had been general improvement since last mentioned date. Respirations were 31; pulse 96. Both knee-jerks were lost. Respiration was easy and normal. The abdomen bulged at inspiration; the abdominal reflexes were good on both sides. The head could be moved rather feebly from side to side and also drawn forward. The sternomastoid was partly restored. The head could not be moved backward. There was no power in the trapezius; all the neck muscles wasted and flabby; all twitch with the galvanic current.

The paralysis of the diaphragm began to improve after the third week and had disappeared by the sixth week. The child improved rapidly in health. A jury-mast support enabled him to support his head. About March 1st the jury-mast support was discarded and the child played about without artificial support to his head. All the muscles of the neck could be moved, although the sternomastoid was still somewhat weak. There was no apparent wasting of any muscle. The course of the disease showed that the myelitis affected a small focus in the cervical cord between the third and sixth cervical nerves, temporarily destroying the function of both phrenic and spinal accessory nerves; the lesion about the phrenic nerve caused a severe but temporary disturbance of respiration. The paralysis of the neck muscles lasted about four months, after which time both sternomastoid and trapezius recovered their power.

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The treatment—protection from chill and respiratory infections, rest, diet, strychnin, electricity—was skillfully carried out by Dr. Edward Villiaume.

ILLUSTRATING CASES.

CASE 1 (Erb).—*High Cervical Poliomyelitis, Reaching the Medulla, Causing Paralysis of the Neck, Diaphragm and Some Cranial Nerves.*—A man, aged eighteen, sickened October 16, 1904, apparently with influenza, having fever, delirium and pain in the neck. The acute stage passed away, leaving the neck so weak that the man could not raise his head. Two weeks later there was total paralysis of both trapezius and sternomastoid muscles; the right arm and especially the triceps was weaker than the left. There was paralysis of the diaphragm more marked on the left side; slight nystagmus, paresis of the abducens; the faradic reactions were lost.

On December 1, 1904, the forty-sixth day of disease, the symptoms of the diaphragm and eyes had disappeared. During the next year a gradual improvement recurred; the head could be raised and moved; the arms and legs were normal; urination and defecation in order; general good health.

On December 5, 1905 (a year later), the neck muscles were atrophied; there was complete destruction of function of both sternomastoid muscles except a small fasciculus of the left, and loss of power of the right trapezius and part of the left. The splenius and levator anguli scapulæ were good but weak. Electric reaction, faradic and galvanic excitability of sternomastoid were gone except in a small fasciculus of the left. A few fibers of the right trapezius and a good part of the left reacted well to faradism. Reaction of degeneration was present. The left rhomboideus was good; the right useless.

In this case the involvement of the cranial nerves was unmistakable. A few weeks after the acute stage there occurred a total atrophic paralysis of both sternomastoid and trapezius muscles, indicating a severe injury to the spinal accessory. Besides this there was also a temporary paralysis of the diaphragm and of the abducens and nystagmus.

Fourteen months afterward Erb found extensive paralysis of the sternomastoid and trapezius muscles. The cranial muscles and diaphragm were normal.

CASE 2 (Wickman*).—*Poliomyelitis with Paralysis of the Dia-*

* Wickman: *Heine-Medinschen Krankheit*, p. 35.

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phragm and Extremities.—A man, aged nineteen, sickened August 10th; he had fever, headache, pain in back and legs. On the third day the patient became weak in the legs, being able to move only the ankles. There was a loss of power in the shoulder. The patient saw double and there was difficulty in swallowing.

August 28th: The patient was seen by Wickman; was improved; had no fever; could not turn in bed or sit up on account of pain in the back. The effort of moving the body caused the abdominal muscles to contract feebly. The muscles of the feet and legs were soft; could scarcely be moved. There was no atrophy in the legs; no patellar reflex. The sensibility was normal. The muscles of the shoulder-girdle were almost useless. The forearms were flexed, especially on the left side; extension was weakened on both sides. Flexion of the hand was good; extension weakened. The deltoid and the extensors of both forearms were atrophied; otherwise the musculature was flabby but not atrophied. Respiration was rapid, superficial, with flattening of nostril. The patient became cyanotic with the slightest exertion, breathing with the accessory muscles of respiration. Breathing was of the costal type; with inspiration the epigastrium and abdomen were drawn in, with expiration the abdomen bulges. There was paralysis of the diaphragm.

The result of the malady as to death or recovery is unfortunately not stated.

CASE 3 (Wickman*).—*Paralysis of Diaphragm and Extremities.*—A man, aged nineteen years, sickened September 19th; had headache, chill, stiff neck, nausea, pain in the legs; during the next two days vomiting and diarrhea. The patient could not support himself on his legs. He had pain in the back; difficulty in urination; constipation. The next day there was complete paralysis of the legs. The left knee was swollen and tender. There was weakness of the arms; enlargement of the spleen.

Nine days later the patient was helpless in bed; the legs, forearm and left deltoid were paralyzed. The abdominal muscles were feeble; there was a weak cough; breathing was almost exclusively costal (paralysis of the diaphragm). The patellar, abdominal and cremasteric reflexes were lost; the sensibility was normal; pupils reacted well, left one smaller than the right; the muscles of the legs and abdomen did not respond to electricity.

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* Wickman: *Heine-Medinschen Krankheit*, p. 83.

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In November complete paralysis of the legs continued. No information as to remaining symptoms could be obtained. As the patient had lived three months it might be assumed that the diaphragm had somewhat recovered its function.

CASE 4 (Collective Investigating Committee†).—*Acute Ascending Poliomyelitis, Involving Phrenic Nerve and Medulla (Landry's Paralysis).*—Child, aged ten, ill five days, with progressive fever, weakness and loss of power in legs and arms until there was complete paralysis in all extremities on the fifth day. Dyspnea was present; tympanitic abdomen; arms and legs were useless; deep reflexes lost. The chest was negative; there was no rigidity of the neck. During examination the patient had two attacks of respiratory failure, revived by artificial respiration. The diaphragm appeared to take no part in respiratory motion.

The patient died on the sixth day; the brain and spinal cord showed the macroscopic appearance of poliomyelitis.

CASE 5 (Wickman‡).—*Isolated Paralysis of Sternomastoid and Trapezius in Poliomyelitis.*—Boy, aged two and one-half, received into the hospital with a pleural effusion, a sequel of measles. When apparently convalescent he became feverish (38.4°C., or 101°F.) and cried at the slightest touch. There was also a noticeable tremor, mostly in the hands and arms, less so in the head. The tremor was fine, apparent in repose, increased when the patient tried to grasp objects. The hands were slightly ataxic; the legs could be moved in bed; patient had difficulty in raising his head from the pillow. While sitting the head could not be held erect. The potassium iodid which the child was taking was stopped. The next day the patient was feverish; the neck muscles were completely paralyzed. If the boy was sitting erect in bed the head fell against the breast and could not be raised; the face was drawn slightly to the left. The knee-jerks, arms, legs and eyes were normal.

After this the fever disappeared and in about two weeks the head could, with some difficulty, be held up. Four weeks later the gait was uncertain and tottering; the head could with effort be held up and moved from side to side. The patient left the hospital in this condition.

Eight years later the patient walked badly and often fell; the right sternomastoid was thinner than the left. The left

† Report of Collective Investigating Committee on Epidemics, New York, p. 68.
‡ Wickman: *Heine-Medinschen Krankheit*, p. 37.

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trapezius was weaker than the right. The knee-jerks were increased. There was strong ankle-clonus; no rigidity. The diagnosis in this case was at first uncertain, the tremor and congestion of the throat being attributed to potassium iodid. The subsequent paralysis of the neck muscles explained the malady.

The weakness of the legs and the increased patellar reflexes and ankle-clonus indicated damage to the pyramidal tracts.

CASE 6 (Henoch*).—*Paralysis of the Neck and All Four Extremities.*—Child, two years and seven months old; suffered first from fever and general distress; later was attacked with paralysis of the neck and all four extremities. After several weeks the paralysis of the neck disappeared, but the paralysis of the arm and legs persisted for three months, so that the child could not grasp objects or leave the bed. Under electrical treatment nearly all disability passed away, so that the child left the clinic with a paralysis only of the left arm and deltoid.

CONCLUSIONS.

From the meager literature of paralysis of the neck and diaphragm in poliomyelitis I would draw the following conclusions:—

(1) Cervical poliomyelitis affecting only the origins of the phrenic and spinal accessory nerves is rare, but in the two recorded cases recovery occurred.

(2) Paralysis of the neck muscles occasionally occurs in poliomyelitis, and except in Landry's paralysis the prognosis is favorable as to restoration of function; some improvement always takes place.

(3) Paralysis of the diaphragm is exceptionally seen in poliomyelitis, and that, although dangerous, recovery is possible.

* Henoch: *Vorlesungen über Kinderkrankheiten*, p. 234.

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THE ORTHOPEDIC TREATMENT OF SPINAL PARALYSIS.

BY PROF. F. LANGE,
Munich, Germany.

The treatment of the acute stage of poliomyelitis has hitherto been exclusively within the domain of internal medicine. Observations which we made in the year 1909 appear, however, to indicate that orthopedics may also participate with success in the treatment of this stage.

We found that children ill with poliomyelitis, and suffering severe pain in the spinal column, became free from pain on the application of a plaster-of-Paris jacket, which embraced the entire trunk exactly like a jacket for spondylitis, and held the spinal column still and at rest.

The suggestion for immobilizing the spinal column of a child in the acute stage of poliomyelitis was first made by Oppenheim. As far as I know, however, orthopedic appliances have not been previously used for this purpose, but the children have been kept flat in bed, or at most supported by pillows to keep them from much moving.

If it is clinically true that the acute stage of poliomyelitis requires the spinal column to be held rigidly and at rest, then it is clear that more is to be accomplished by careful fixation in an orthopedic jacket or an orthopedic bed than by simply lying in bed.

For we must try, when a child is acutely ill with poliomyelitis, to prevent and limit, as far as possible, the extension of the inflammatory process in the spinal cord.

I myself think that at present we can rely less on internal remedies or on hydrotherapeutic measures than on orthopedic fixation of the spinal column. All the experience we have had in other acute inflammations indicates that the keeping of the affected parts at rest is the most important requirement for a speedy recovery.

I should like, therefore, in your country, where in recent years you have been visited by such epidemics of poliomyelitis, to earnestly recommend in the acute stage the immediate fixation of the trunk in an orthopedic bed or by a plaster-of-Paris jacket.

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When the acute stage is passed and the losses which the inflammation in the spinal column has produced can be reviewed, namely, the inability of the patient to move, then a second task devolves upon the orthopedist, which is to assist, as far as possible, the restoration of the paralyzed muscles and to prevent the occurrence of the contractions. For a year after the beginning of the illness the possibility still exists that the paralyzed muscle will recover and therefore, we must do all we can in the first year to assist this recovery.

It is well known that we can favorably influence the nourishment of an affected muscle and the circulation of the blood in it by the application of electric currents, by massage, and by air and water baths of the most varied kinds. I have nothing new to tell you about that, only I should like to warn you against a rough, hard massage and, above all, against a deep, firm, stroking massage. When you reflect how soft and tender the muscle paralyzed by poliomyelitis is, you will acknowledge that we must be very careful in handling such a delicate structure. Just as it is possible by a powerful massage to remove fat on an undesirable part of the body, so can we, I am convinced, weaken and injure a paralyzed muscle by a rough massage, instead of strengthening it, as is our object and intention.

The prevention of contractions makes the application of orthopedic apparatus necessary, but we must be careful not to overdo a good thing. Normal muscle may be injured and weakened, as we see every day, by the pressure of any orthopedic apparatus and consequently fall into a partial atrophy. The paralyzed muscle is still more sensitive to pressure than is the healthy, and, therefore, we must limit the application of orthopedic splints as much as possible both with respect to the length of the time they are worn and to the weight of the apparatus. My method will best be made clear by some examples.

If I have determined in a little patient with poliomyelitis that there is a paralysis of the tibialis anticus and posticus, then I know there is danger that in a short time, though using the lame foot, a bad pronated flatfoot (pes planovalgus) will occur. To prevent the occurrence of such a deformity I do not use a large apparatus, as, for instance, a Hessing splint, for it is precisely these forms of apparatus which cause the greatest atrophy of the muscle, but I content myself with a celluloid support, reinforced with steel wires, which brings the foot into a relative supinated position and

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prevents the sinking of the arch of the foot. For use during the night I order an internal splint of similar constitution, which fixes the foot in a stronger supinated position and relieves a shortening of the pronating muscles which possibly may have taken place during the day; this stiff shell is without lacing and avoids all pressure on the paralyzed muscles.

I proceed in a similar manner in paralysis of the other muscles, the extensor digitorum and the peronei. Here there is danger that a club-foot will occur; to prevent this I have used during the day a support similarly made of celluloid, reinforced with steel wire, which presents an oblique plane sloping from without inward, and which with every step inclines the foot toward a pronated position. During the night I employ an outside splint of similar constitution which holds the foot in a pronated position.

In a case of paralysis of the dorsal flexors I have used during the night a splint which forces the foot into an extreme pes talus position and powerfully stretches the Achilles tendon, which is strongly inclined to contract. During the day I also avoid cumbersome apparatus with these children. I restrict myself to putting a small splint into the back of the shoe, which prevents the sinking of the foot beyond 90° and thereby makes the over-extension of the paralyzed dorsal flexors impossible.

If the gastrocnemius is paralyzed I take care that the foot is fixed in an extreme equinus position during the night. For the day I employ only a plate, which brings the foot into a slightly supinated position and thereby counteracts the inclination to pronation.

In paralysis of the quadriceps I also avoid apparatus during the day as far as possible. If the children are able to walk notwithstanding their paralysis, I simply let them use their legs without any artificial support. If there is a tendency to deformation of the knee, whether it be a genu valgum or a recurvatum, I restrict myself to the use of a night splint which fixes the leg in the opposite position. Only when the children are unable to walk without an apparatus do I support the paralyzed limb with either a short celluloid splint, or with the usual steel truss-belt apparatus. I also avoid here the laced leather case, because it causes a much greater atrophy of the muscles than the old belt apparatus.

If you begin early with the orthopedic treatment and carry it out consistently you will help the patients very much, and you will, as a rule, be able to prevent contractions. Even in case of

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paralysis of the tibialis anticus and posticus, where the burden of the weight of the body extraordinarily favors the formation of a pronated flatfoot, you can by these means certainly prevent the formation of a deformity, as I know from many years' experience.

There are patients who are so well satisfied with the result of this simple treatment that they do not want anything further and prefer to continue it during their lives rather than to submit to an operation. The majority of the sufferers, however, sooner or later want to be relieved, if possible, from the apparatus which makes them dependent upon the doctor and the instrument-maker. For this purpose we have several procedures: Redressment with or without tenotomy, transplantation of the nerves, arthrodesis, and transplantation of the tendons.

Redressment is always necessary as a preliminary operation when the patient first comes under treatment, when a contraction has taken place. On this point all the authors agree. Lorenz, in Vienna, goes still further. He thinks that in most cases a redressment is sufficient and that in a paralytic club-foot nothing further is necessary than tenotomy, redressment, and a plaster-of-Paris cast for two months.

My experience in this particular direction, especially since the era of tendon transplantation, is to the contrary. In most paralytic deformities, if you restrict yourself to redressment, you get sooner or later a relapse of the deformity. An exception to this is seen in those deformities, above all certain bad cases of equinus, in which single muscles which were temporarily paralyzed by poliomyelitis recover in a quarter of a year or in half a year after the illness; notwithstanding this they are generally not able to raise the foot, because in the meantime a severe equinus and such a stiff contraction of the Achilles tendon have developed that the overspanned and weakened dorsal flexors are powerless against them. However, if you remove these difficulties by tenotomy and redressment and give the paralyzed dorsal flexors an opportunity to shrink and contract by fixing the foot for a sufficient time in a pes talus position, they can afterward raise the foot again in an approximately normal way. In such cases I do not transplant the tendons, but restrict myself to redressment. I think, however, that this method of treatment, according to Lorenz's recommendation, will not be successful, as a rule, in cases of paralytic deformities, but rather that it will generally be necessary to repair the loss of the paralyzed muscle by a second operation.

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Three methods are at our disposal. I shall begin with the newest operation, nerve plastic.

If we had the same results with our poliomyelitic patients in nerve plastic as we have in our experiments on animals, then nerve plastic would without doubt be the operation which would best solve the problem of conducting new power to the paralyzed muscles. That nerve plastic can achieve good results in men is proved by observation in cases of facial paralysis, but a result entirely free from objection in the case of a poliomyelitic patient I have myself not yet seen.

If the muscles are given an opportunity to shrink, whether it be by operative shortening or by a long application of connecting apparatus or bandages, then the result attained may be due to those measures and the transplantation of the nerves may perhaps have been an entirely superfluous operation. I have, as I have said, neither among my own patients nor among those operated on by other physicians so far seen undoubted success. I also think that those peripheral paralyses in which there soon occurs a degenerative reaction and the destruction of the nerve-end apparatus in the muscle, and by which finally the whole muscle suffers more or less fatty degeneration, are not proper subjects for nerve plastic. Nerve plastic in a degenerated muscle is always without result, as its warmest advocate, my colleague Spitzy, acknowledges. If you resort to nerve plastic in a case of spinal paralysis before degeneration has set in and see later that the muscle works, it is still no proof that the nerve plastic has been of service, for in the first months a paralysis can spontaneously disappear.

These observations and reflections make me at present still reserved with respect to nerve plastic. I should be very glad if we could attain in this way an ideal cure of our paralyzed patients, but the results hitherto achieved unfortunately do not seem to furnish sufficient ground for such a hope.

I now turn to arthrodesis. It is unpleasant to be obliged to stiffen a joint, to sacrifice a function of the body. Circumstances, however, often compel us to do it and when by such an operation we attain a greater ability to walk, we may not ask whether we like the operation or not. In case of the complete paralysis of the muscle of the lower leg and foot arthrodesis can be a great blessing, because the instability of the talo-crural and talo-calcaneus joint, and in severe cases also of the joints of the fore part

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of the foot, is done away with. In cases of this character I perform an arthrodesis of the foot. As a rule, however, I do not operate before the fourteenth year, as too early operation reduces the prospect of bony union of the joint surfaces and favors a relapse of the deformity.

The fixation of the foot by the operation greatly facilitates walking on level, smooth ground, but the patients are often very much hindered by the stiffness in walking on uneven, hilly ground, because the stiff foot cannot adapt itself to the irregularities of the surface of the ground; the success of the operation is thus impaired. In the room the patients can walk better with their arthrodesed feet than before with tottering ones. Out of doors and in walking for a long time the foot after the arthrodesis often gives out sooner than before the operation. This experience induces me to seek another operation which would retain the movement of the foot to 90° and only make the dangerous and annoying sinking of the point of the foot beyond 90° impossible.

At first I sought to attain this by puckering and shortening the dorsal flexors. The success of this method is at first very good, but according to my experience does not yield permanent results in children; only in adults have I seen permanent results from shortening of the dorsal flexors. In children I have, therefore, given up the shortening of the paralyzed dorsal flexors and instead use silk to prevent the sinking of the point of the foot below 90°. The method, which I first used in the year 1903, creates in certain respects *artificial joint ligaments out of silk.* The technic is as follows: I make four small incisions—two on the median side, one over the navicular bone and the other over the tibia, 5 cm. above the joint, and two on the lateral side over the cuboid and on the fibula. I carry from the tibia to the navicular bone and from the fibula to the cuboid six to eight strong silk threads which are very securely sewed to the periosteum above and below and are passed in the middle through the deep adipose tissue over the capsule of the joint. The results of the operation are good and permanent if careful after-treatment is carried out for a year.

If all the muscles are not paralyzed, but some strong ones are well conserved, *transplantation of the tendon* takes the place of arthrodesis. The ingenious idea of using living muscles in the place of paralyzed ones in order to restore a lost function was carried out for the first time by Nicoladoni in the year 1880, but

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his results are said not to have been good. The surgeons who themselves collaborated in the development of the method understand that very well, for the difficulties which the new method had to overcome were great. When I adopted the Nicoladonian idea in the year 1897, I was not spared disappointments. One cause of my failure, as I was able to establish by supplementary operations, was the subsequent stretching of the paralyzed, flabby and tender tendons. This induced me in the year 1898 to abandon the Nicoladonian method, in which the belly of the invigorating healthy muscle is transplanted into the paralyzed tendon, and in its place to sew the invigorating muscle directly into the periosteum. As to whether the old Nicoladonian transplantation or the new periosteal method is the more advantageous has been for many years much disputed in Germany. To-day the periosteal method is probably preferred by most orthopedists. The census of the American physicians, which was taken by my highly esteemed friend Lovett in Boston, was of decisive value in determining the opinions here. Out of thirty-three physicians, twenty-eight declared themselves in favor of the periosteal method and only five for the old Nicoladonian transplantation. It is thereby probably sufficiently proved that in general the periosteal method is preferred. I do not mean by that that you should always transplant directly the periosteum. Now and then I myself make use of the paralyzed tendon, if it is very strong, as, for instance, the Achilles tendon, and sew the belly of the invigorating muscle directly into it, but the periosteal transplantation has become the rule with me and probably with other orthopedists. I use an artificial tendon made of silk to lengthen the muscle. The method of using artificial tendons was first carried out in the year 1875 by Auger, who used a piece of this material to supply the loss of a tendon. Gluck, in Berlin, then demonstrated by numerous experiments on animals that you could cause artificial tendons made of catgut to heal in and that catgut is replaced in the body by connective tissue. Gluck has also made successful experiments of this kind on men. His reports were not received with the confidence they deserved.

When I, in the year 1899, for the first time introduced a silk tendon in an operation for the transplantation of a quadriceps, I was not free from apprehension that the silk tendon would finally be expelled. My experience with men and numerous experiments on animals demonstrated to me that my fears were without

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foundation. Allow me to go a little further into the matter of artificial tendons.

That the most painstaking asepsis in the operation is under all circumstances necessary is a matter of course. Tendons which have been boiled only in pure water heal up best. Such a silk tendon is covered with a capsule of connective tissue in a few weeks without the manifestation of any pronounced inflammation. The permanent results of these silk tendons boiled in water are not good. If the human body contracts an infection and bacteria circulation in the blood, then the bacteria settle with predilection in these silk strings and cause a suppuration which renders the removal of the silk threads necessary. This experience with silk threads which have been used for tying has often been made. Not infrequently such a thread abscess appears many years after the operation and leads to the expulsion of the silk. This experience shows that the silk thread must be saturated with some antiseptic, if it is to be permanently secure against an attack of bacteria. I proceeded, therefore, to boil the silk in a solution of sublimate, one to one thousand, before the transplantation. The sublimate, as was established, enters into an intimate, extraordinarily permanent chemical union with the silk and seems to permanently protect it against a later infection by bacteria which circulate in the blood and to prevent the formation of a subsequent thread abscess. I have at least never as yet experienced a secondary infection, among the many hundreds of silk tendons, in a tendon that has been healed in for several months and has functionated. The sublimate silk protects us indeed from a subsequent infection, but it has a disadvantage: healing takes place with much more difficulty than when the silk is boiled in pure water. If you investigate the healing process with sublimate of silk in experiments on animals you will find that the sublimate silk ten days after its insertion causes a collection of secretion in its immediate neighborhood. This pus-like secretion is entirely free from bacteria, as the bacteriologic examination shows, and arises without doubt from the chemical irritation which the sublimate exercises on the leukocytes. This aseptic secretion is, as a rule, reabsorbed in animals, and finally a distinct capsule is formed in the immediate vicinity of the silk. In men, according to my experiments, the reabsorption of this pus-like secretion does not take place so well as in animals. We must enable this secretion to escape. For this purpose I drain the places in which I have

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inserted silk tendons for two days with a wick of sublimate gauze. The wounds which show a secretion must then be carefully dressed, often for two weeks, because otherwise an infection of the originally aseptic secretion and thereby also of the silk can easily take place. By this the healing of the silk boiled in a sublimate is made very difficult and tedious. This induced me to seek a thread that would heal as easily as the silk boiled in water and be as secure against subsequent infections as the sublimate silk. By experiments on animals, carried out by my assistant, Dr. Frey, I was able to establish that these requirements are best fulfilled by a paraffin-sublimate silk. This silk you obtain by cooking the silk, which has been boiled in sublimate and then well dried for two days under aseptic precautions, for an hour in paraffiin (melting point, 60°), in a water bath; the paraffin makes the direct contact of the sublimate with the fluids of the tissues difficult, as we are also able to establish by experiments on animals, and thereby prevents the occurrence of the pus-like secretion. I have used this paraffin-sublimate silk for five years exclusively for my artificial tendons, and I can recommend it to you most highly with the greatest conscientiousness as the result of my favorable experience. If, notwithstanding the employment of this silk, a silk tendon does not help, you may assume either that you have not proceeded properly in the preparation of the silk, or that a mistake has been made in the technic of the operation.

I have gone particularly into the introduction of the artificial tendons because I think that silk is employed much too little in surgery. Above all, it is much too little recognized that silk which is functionally employed is penetrated and surrounded with a very firm and thick connective tissue. At the time of removal of the plaster-of-Paris bandages from patients on whom I have operated two months before, the artificial tendons can be felt to be only as thick as immediately after the operation. When, however, the silk tendon has worked for some time it becomes thicker and thicker. Finally, the artificial tendons attain, for instance, in a quadriceps plastic, in which they are originally only as thick as a knitting needle, the thickness of your little finger. Thereby the extremely interesting condition was found that a thick covering of true tendon tissue had penetrated into the interior of the silk and was thoroughly grown together with it. This encapsulation with true tendon tissue guarantees the durability of the arti-

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ficial tendons and shows that we can cultivate a firm connective tissue in places where we need it.

The transplantation into the periosteum and the silk tendons are not alone sufficient to obtain entire success in a tendon transplantation, but they must be assisted by a proper plan of operation. About this there was also a dispute among the German orthopedists for many years. Vulpius wanted to replace every muscle that had been lost. In order to accomplish this, he divided the sound muscle into two or three parts and transplanted these parts hither and thither. Thereby the plan of the operation became extraordinarily complicated. The operation did not fulfil the expectations of the operator, for the separated parts of the muscle did not learn to work independently but always contracted simultaneously with the original muscle, and it was, therefore, not possible in this way to again restore the functions of the paralyzed muscles.

I, therefore, adopted the principle that we must sacrifice unimportant functions, and that only the most important movements should be re-established. If you go about it properly you can transplant important muscles and can leave out of the question the complicated and impracticable division of the muscle. The plan of operation is thereby extremely simplified. It is sufficient on the foot if I have three healthy muscles at my disposition; these I divide so that I have one muscle as plantar flexor, which I allow to draw on the calcaneus at the normal point of attachment of the tendo Achillis. The other two muscles extend as dorsal flexors on the front of the lower leg and the foot. The muscle which simultaneously supinates must be sewed into the navicular bone and the pronating flexor into the cuboid.

By the extension of the muscle it is generally indifferent how the upper central half of the muscle extends: it may describe a curve or another irregular crooked line. The direction of the lower peripheral half is decisive for the working. An example will best illustrate this. If I wish to use the peroneus longus as a substitute for the tibialis anticus, I cannot conduct it in a straight line to the navicular bone. The new muscle will thereby indeed receive dorsal flexor attributes, but will not supinate the foot as the lost tibialis anticus did, but it will pronate it and draw it over into a pronated position. In order that the new muscle may supinate it must have in its lower half exactly the same direction as the tibialis anticus.

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Finally, you must lay weight on a careful supplementary treatment with night splints and supports. You cannot, in the beginning, after the removal of the bandage, exact too much from the muscle, or else it may stretch or tear. On this point there is no difference among the orthopedists, and therefore I leave this point. All that I have here described I have followed out for years and notwithstanding my results were very varied. I succeeded, it is true, by the transplantation of the tendons in preventing a relapse of the deformity which took place so often in simple tenotomy and redressment, and I also had the satisfaction that the invalids learned to walk without apparatus, or, at most, with a small splint. But I did not regularly accomplish a complete re-establishment of the function. The function of the transplanted muscle often did not return or remained weak; this I have especially observed in the foot. In the transplantation of the quadriceps the restoration of the function is much more complete. This is singular, for the work which is necessary for the movement of the knee-joint is much greater than that for the movement of the foot. What I was still more astonished at was the extraordinary function I obtained when I substituted the vastus externus for the gluteus medius and minimus. In this operation I detached the upper attachment of the vastus externus from the trochanter major, wove eight or ten silk threads into it and sewed these into the crista iliaca. By this operation the patients receive the power of adduction of the leg in a most normal way. These observations showed that the want of success in the transplantation in the foot must have a distinct cause. I therefore examined more carefully a number of patients in whom the functions of the transplanted muscles had not been restored. The transplanted tendon had united with tendonous adhesions very firmly to the bones, ligaments and fascia, and thereby every movement of the transplanted tendon, and with it of the foot, had become impossible. If, nothwithstanding these attachments, the patients were saved from a relapse, this occurred because the attached tendon had fixed the foot in the corrected position exactly as in a tenodesis. How, then, were these attachments to be avoided? I accomplished it by boring a channel for the muscle directly through the adipose tissue, so that the transplanted muscle was always separated by a layer of fat from the fascia and the other tissues. In this way the attachment between the tendons and the bones or the fascia was avoided. Since I have done

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this I have no longer to complain of insufficient function of the transplanted muscles.

If you take into consideration the great difficulties which the development of the method of operating in transplantation of tendons has undergone, you will be able to understand and properly appreciate that in recent years many objections have been made to the operation. At the Paris Congress of Surgeons, in the year 1907, most of the surgeons were inclined to arthrodesis and condemned tendon transplantation. But when you hear that the first chairman of the committee on this subject, Dr. Kirmission, of Paris, had only performed fifteen tendon transplantations, and expressed his disinclination for this operation after so little experience, it does not weigh much in the scale. I can also explain the unfavorable inclination which Lorenz has to the transplantation of tendons only on the ground that he has formed his opinion from a small number of operations.

We have all, perhaps, in the beginning, in the first burst of enthusiasm, performed too many tendon transplantations. We have thereby learned to distinguish more sharply between the indications for the transplantation of tendons and arthrodesis. We shall also still learn, I have no doubt, to improve the results of our tendon transplantations. The transplantation of tendons has already brought endless blessing to our cripples and it may with justice be counted among the greatest advances that orthopedic surgery has made in the last decade.

NOTE.—The foregoing papers by Drs. Holt, Flexner, Snow and Lange were presented in a symposium on poliomyelitis at a joint session of the American Pediatric Society and the American Orthopedic Association. In addition to these, there were papers by Dr. I. Straus, of New York, and Dr. Robert W. Lovett, of Boston. Both of these papers have been published elsewhere. "The Pathology of Acute Poliomyelitis, and Its Bearing upon the Symptoms of the Disease," by I. Straus, A.M., M.D., appeared in PEDIATRICS for August, 1910. "The Occurrence of Infantile Paralysis in Massachusetts in 1909," by Robert W. Lovett, M.D., was published in 1910 as a Report of the Massachusetts State Board of Health.

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DISCUSSION OF POLIOMYELITIS.

Discussion on these papers was opened by Dr. Bernard Sachs, of New York, who said that it was a very difficult matter to open a discussion on a question that had been so thoroughly talked over as this, and that he had not had the privilege of hearing all the papers, and might repeat something already said. Inasmuch, however, as the medical profession in New York had been thoroughly saturated with the subject since 1907, he thought that he might refer to one or two points of general interest.

In the first place, he wished to say that he did not know what the readers of the papers had expressed; but he hoped that one of two names only would be retained for this disease, the proper designation of which is infantile spinal paralysis. He trusted that the term infantile paralysis would not be allowed to go down as the term favored by these societies; because he considered infantile spinal paralysis the proper desigation, in contra-distinction to infantile cerebral, or spastic. While they had been speaking of encephalitis, or polioencephalitis, he thought that the proper designation of this would be polioencephalomyelitis. He contended that nothing had occurred in any epidemic to propagate the claim that the infantile spastic paralysis is the counterpart of the infantile spinal paralysis, so few actual cortical lesions having been found that he could safely say that this contention of Strümpell had not been sustained. The encephalitis is of the lower brain axis. The infantile spastic paralysis that Strümpell had in mind would necessarily be of cortical origin; but in the epidemics described in Europe and in America, there have been no cerebral cases of any account—only one or two, and these imperfectly described, having been the counterpart of the infantile spinal cases. While in all text-books on the subject, this view of Strümpell seems to be followed, Dr. Sachs wished to state in the presence of the pediatrists and the orthopedists that there had been no evidence of the truth of this theory furnished. He thought that it would be a good thing to drop it until such proof should be advanced.

He also thought that although some pathologic conditions show that there is much more than a mere anterior poliomyelitis present in this disease, it would be well to retain that designation, which is so well known, as the lesion is so largely in the gray matter. Physicians do not drop the designation of posterior spinal sclerosis or of tabes dorsalis, because in locomotor ataxia there is more than involvement of the posterior roots.

Passing from this question, he said that he wished to go on to just one other that he considered to be of importance, and which he believed that not too much had been said on. This was

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the question of treatment. He felt that he could hardly venture to say anything on this subject, after the presentation of Professor Lange, but he desired simply to add that he had a perfect horror of the time wasted by the majority of physicians in the treatment of these cases of infantile spinal paralysis. He considered the time given to massage and electricity to be practically wasted. He would not say that this method of treatment does harm, but he could say that in his experience he could not put his finger upon any definite good that had been done by either massage or electricity. So, in the presence of the orthopedic surgeons, he wanted to say a word of encouragement to them in insisting upon their rights in the matter. He believed that more good would be done by the early institution of orthopedic measures than by the use of electricity and massage. While he could not quite agree with Professor Lange in ascribing direct harm to massage in these muscles, he did believe that the harm done by it was in wasting time, and he trusted that the bloodless and the bloody surgery would be applied. He thought that the earlier this was done the better the results would be.

He wished also to say, in connection with the subject of epidemic poliomyelitis, which the New York committee had taken carefully into consideration, that they concluded that the so-called atypical forms of this disease are so rare, as compared with the typical, that one should not allow one's conception of the disease to be disturbed by the occurrence of atypical cases now and then.

Dr. H. Winnett Orr, of Lincoln, Neb., hoped that he would be pardoned for saying a word in this discussion, because he was anxious to bring down the death rate in the Nebraska epidemics of poliomyelitis. He had been endeavoring to collect as much information as possible regarding the epidemic that had occurred in that State, and he trusted that he would be forgiven for stating that he had done this practically single-handed. For this reason he was unable to get as much information as he had hoped to obtain. There were not 200 cases, as had been stated, but 600. Dr. Orr had reports of 619 cases, concerning two-thirds of which he had been able to get detailed information. This information he had summarized and placed on a little chart. This was included in a statistical table of cases treated at the Nebraska State Orthopedic Hospital, which had been printed, which he distributed among those present without further comment, although he said that he might, if he had the time, say a good deal more about these cases.

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AMYOTONIA CONGENITA (MYATONIA CONGENITA OF OPPENHEIM).

BY J. P. CROZER GRIFFITH, M.D.,
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About ten years ago Oppenheim, of Berlin, was impressed by a symptom-complex which he had observed in a number of instances. In his first contribution to the subject (*Monatschr. f. Psychiat. u. Neurol.*, 1900, Vol. VIII., p. 232) he describes the symptoms, although he does not detail the cases. From a later publication (*Berlin. klin. Woch.*, 1904, Vol. XLI., p. 255) we gather that these were probably 4 in number. As described by him the disease consisted of a flaccid palsy appearing to be congenital, most marked in the extremities, especially the lower, and varying in degree up to complete paralysis. There was no actual atrophy; the sensation was not disturbed; the tendon reflexes were abolished; the electrical contractility much diminished; the hypotonia so great that there was abnormal passive mobility at the joints. To this condition he gave the title "myatonia congenita." As this is unfortunately so similar to "myotonia congenita," or Thomsen's disease, that constant confusion arises, various other names have been proposed, that of *amyotonia congenita* being, probably, as good as any.

A number of cases have been reported since Oppenheim's publication. Tobler (*Jahrb. f. Kinderhk.*, 1907, Vol. LXVI., p. 33), writing in 1907, published abstracts of those recorded and made a careful analytical study of them. His contribution was based upon 17 instances of the disease, viz., Oppenheim 5 cases, Berti 2, Sorgente 2, Muggia, Spiller, Kundt, Iovane, Comby, Rosenberg, Bing and Tobler, each 1 case. Collier and Wilson (*Brain*, 1908, Vol. XXXI., p. 26) give the abstracts of 25 cases, not including the earlier ones reported by Oppenheim. They omit the 2 reported by Sorgente on the ground that they differed too much from the type, as also the case of Muggia. They add 14 cases to those reported by Tobler, viz., Batten 3, Collier and Wilson 2, Schüller, Cattaneo, Coombs, Lugenbühl, Beevor, Variot and Devillers, Comby, Leclerc, and Whait each 1 case.

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I have had the opportunity of observing the following very typical instance of the disease:—

Charles K., white, aged fifteen months, admitted to the Children's Medical Ward of the Hospital of the University of Pennsylvania, December 8, 1909, on account of paralysis of the limbs, which had been diagnosed elsewhere as poliomyelitis. The father was a heavy drinker, the mother a drunkard, and both, especially the latter, appeared to be mentally below normal. The 6 other children of the family were badly neglected, but none of them exhibited any condition at all similar to that of the patient. The mother stated that the child had never suffered from any acute disease, but had never been able to use his legs in any way or to handle objects; he took slight notice of anything, cried very little, and had not learned to talk at all. He had never been able to sit up.

In answer to a communication from me, the physician in attendance at his birth stated that the infant had seemed normal at that time. He had not seen the family since. On examination when admitted the following notes were made: The child is well nourished, but pale, without evidence of rickets. The movements of the face and eyes are normal throughout; the thoracic expansion is diminished, the respiration being largely abdominal; the legs are motionless and flaccid, the feet in extension in the position of foot-drop, and outwardly rotated; the muscles of the legs are soft, but without actual discoverable atrophy; the child can move the toes and legs very freely when stuck with a pin. Passive movement at the knee-joint is unusually free, but there is resistance (contracture) at the ankle-joint. The muscles of the arm are soft but not atrophic; the hands are held in a position suggesting tetany, and there is slight resistance to passive movements of the fingers and wrists. Sometimes the fingers are spread, as in athetosis. There is a curious dimpling of the dorsal surface of the fingers at the junction of the proximal and second phalanges, and also, to a less extent, at the metacarpo-phalangeal articulations. (Fig. 1.) The child can move the arms very feebly and slowly, with little successful effort to handle objects. There are no tendon reflexes obtainable; the abdominal reflex is absent; the cremasteric present. The child is entirely unable to sit unsupported, and when held in this position extreme kyphosis develops and the head drops in any direction in which gravity acts upon it. When released the trunk falls in a similar manner,

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sometimes forward, with the head against the ankles and the arms lying helplessly where they happen to be at the time, and he is entirely unable to change this position. (Fig. 2.) The child is apathetic, takes little notice of its surroundings, cries little, and, in fact, appears to be somewhat deficient mentally.

Examination of the blood gave hemoglobin, 60 per cent.; red blood corpuscles, 5,210,000; leukocytes, 10,900. The urine was normal.

An electrical examination kindly made by Prof. Charles K. Mills showed an entire absence of contractility with rather strong currents, both faradic and galvanic. There was no reaction of degeneration.

Treatment with massage, faradic electricity and strychnin was commenced and efforts were made to awaken the child's intellectual powers. Improvement, although slow, has been positive. A note made February 18th, 1910, about two and a half months after admission, reads as follows: The intellectual power has improved greatly, the child smiling and taking notice of the nurses, and making efforts to repeat words after them. The power in the arms has increased, and the infant now can grasp a biscuit and eat it unaided. All movements of the arms are, however, slow and feeble. There is also slight increase of movement in the legs. Under electrical or other stimulation there is a decided increase of movement in the extremities. It is to be noted, however, that the application of quite strong currents seems to give no pain, and no distinct muscular contraction has yet been obtained by electricity. The abdomen is distended and the abdominal reflex still absent. The child lies with the thighs in outward rotation, the knees flexed, the feet in plantar flexion. There is slight increase in the passive mobility of the ankle-joints and of passive rotation of the knee and hip-joints. This permits the soles of the feet to be turned directly upward with ease. Passive abduction at the thigh is not increased, and there is no abnormal mobility of the arms. The shoulders, however, are abnormally mobile, and lifting the infant by the arm-pits brings the acromial process beyond the pinnæ of the ears. (Fig. 3.)

Examination by Dr. J. W. McConnell on this date still showed absence of faradic contractility with large currents.

The notes made by me on April 26, 1910, four and a half months after admission, read as follows: Has had more or less constant temperature from normal to 101°F.; intelligence seems

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nearly normal and he is rapidly learning to talk. When propped upright he can hold head erect; is not able to turn himself in bed. Respiration is still chiefly abdominal; no abdominal reflex. Hands have improved slightly, but can do little more than put a biscuit to his mouth; very little power in legs. Slight contracture of hamstrings. Has passed several ascarides at times under treatment, but ova can no longer be found in the stools. Looks pale and has lost some weight, yet seems bright. Blood gave hemoglobin 50 per cent., red corpuscles 4,630,000, and leukocytes 8,000. Urine normal.

Inasmuch as comparatively full reports of the various published cases have been given in the collective studies of Tobler, Collier and Wilson and others, I have contented myself with abstracting in very brief form, yet with the essential features, the clinical histories of these cases, adding to them, likewise in condensed form, the accounts of 16 others, which I have found in journal literature, not included in either of these series. These are, namely, the cases of Simonini, Wynter, Ausset, Thompson, Brunard, Gastonguay, Hartenberg, Orbison, Silvestri, Lereboullet and Baudouin, Collier and Holmes, Laffer, and Schlivek, each 1 case, and Haberman 3 cases. These, with my own, make the total number equal 49, besides a case of Reyher and Helmholz, of which I have been unable to find any clinical details, although I have the report of the autopsy. The histories are arranged as far as possible in the chronologic order of their publication. Some of the cases are of doubtful diagnosis, and time will probably eliminate them from this category. I have consulted the original publication in every instance in the list except that of Berti, which was not accessible.

It should be stated that a case with somewhat similar symptoms has been reported by Bernhardt (*Neurolog. Centralbl.*, 1907, No. 1), but is excluded by Tobler, and similarly one by Beevor (*Brain*, 1902, Vol. XXV., p. 85) excluded by Collier and Wilson; in both instances, it seems to me, with sufficient reason, and I have not placed them among the abstracts given. A case published by Sevestre (*Bull. Soc. Ped.*, February 21, 1899, p. 3) exhibited reaction of degeneration and decided muscular atrophy and cannot properly be called amyotonia; and Hutinel (*Ibid*, p. 9), in discussing this case, reported briefly 2 of his own, which must be excluded for similar reasons.

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1-4. Oppenheim (*Monats. f. Psychiat. u. Neurolog.*, 1900, Vol. VIII., p. 232.)

No separate details of the cases are published. An abstract of his description of the symptoms of the disease in general is given on the first page of my article.

5. Batten (*Brain*, 1903, Vol. XXVI., p. 147).

Boy, six years; family history negative; dated from birth. No marked wasting, but limbs thin; all movements possible but weak; face normal, except somewhat expressionless; knee-jerks absent; contracture of ham-strings; reaction only to strong faradic current. Died of intercurrent disease.

7. Batten (third case) (*Ibid*).

Girl, six years; family history negative; dated from birth. Cannot sit or stand; no local wasting, but muscles small and limbs thin; can use hands imperfectly; slight contracture at knee and hip-joints; knee-jerks absent; intelligence normal. Died of intercurrent disease.

8. Muggia (*La Pediatria*, 1903, Vol. XI., p. 179).

Girl, four months; family history negative; dated from birth. Complete flaccid paralysis of all four limbs; knee-jerks present, perhaps a little weak; faradic and galvanic contractility normal. Died of bronchitis.

Case not included in Collier and Wilson's list. Anomalous in condition of the knee-jerks and electrical reactions.

9. Oppenheim (fifth case) (*Berlin. klin. Woch.*, 1904, Vol. XLI., p. 255).

Nineteen months; family history negative; dated soon after birth; has gradually improved; moved arms at nine months. *Examination.*—Lower extremities nearly motionless, except feet and toes; muscles doughy but not atrophic; knee-jerks absent; almost no reaction with strong electrical currents; face normal; no longer much diminution in strength of arms.

10. Schüller (*Wien. klin. Woch.*, 1904, Vol. XVII., p. 722.)

Boy, nineteen months; always weak; learned to stand and sit, but grew worse, and at ten months no longer able to stand and sat with difficulty. *Examination.*—Can move legs slightly, cannot stand or sit; is markedly hypotonic; no atrophy; knee-jerks absent; great diminution of electrical contractility.

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11. Spiller (*University of Pennsylvania Medical Bulletin,* 1905, Vol. XVII., p. 342).

Boy, twenty-two months; family history negative; probably dated from birth. All movements of limbs possible, but feeble; muscles soft but not atrophic; knee-jerks absent; sits and supports head poorly; cannot stand; strabismus and possibly amaurosis; swallowing difficult and nasal tube feeding necessary; increased passive mobility; faradic contractility present. Died of acute febrile condition.

12. Berti (Quoted by Tobler, q. v.).

Boy, three days; family history negative. Flaccid paralysis of arms and legs, with complete hypotonia; respiration diaphragmatic; knee-jerks absent; faradic contractility uncertain. Improved very slightly in three weeks.

13. Berti (second case) (*Ibid*).

Girl, five years; family history negative; dated from birth, when had complete flaccid paralysis of arms and legs and somewhat of the neck. Very slowly improved and then rapidly when nearly five years. *Examination.*—All movements possible, but feeble and slow; muscles soft; gait unsteady; knee-jerks diminished; faradic contractility diminished; intellect normal.

14. Kundt (*Inaug. Dissert. Leipsig,* 1905).

Girl, eighteen months; family history negative; dated from birth, when had no movements of feet at all; gradually improved. *Examination.*—Arms seem normal; can sit; movements possible in feet and toes and slightly in the neck; passive hypermobility; muscles soft and flabby; face normal; knee-jerks absent; faradic contractility diminished; no reaction of degeneration; possibly slight contracture; intellect normal. Improved slightly under treatment.

15. Iovane (*La Pediatria,* 1906, Vol. XIV., p. 190).

Boy, three months; family history negative; dated from birth. Complete flaccid paralysis of legs and almost complete of arms; cannot hold head erect; muscles of trunk atonic; shallow thoracic respiration; no atrophy of muscles; face normal; knee-jerks absent; galvanic contractility diminished and faradic still more so. Died of bronchopneumonia at five months, unimproved.

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16. Sorgente (*La Pediatria,* 1906, Vol. XIV., p. 358).

Boy, twenty-seven days; sister had same disease; no fetal movements; dated from birth. *Examination.*—Complete flaccid paralysis of upper and lower extremities; muscles flabby; can move head very slightly; thoracic respiration absent; knee-jerks absent; faradic and galvanic contractility greatly reduced; no reaction of degeneration. Improved slowly for ten days, then died of convulsions.

17. Sorgente (second case) (*Ibid*).

Sister of previous case; five days old; no fetal movements. Complete flaccid paralysis of legs; nearly complete of arms; other symptoms as in the first case. Death from convulsions.

Both of these cases are excluded by Collier and Wilson on the ground of family history.

18. Cattaneo (*La Clinica Mod.,* 1906, Vol. XI., p. 282).

Girl, four months; dated from birth; no fetal movements. Complete hypotonia with only slight movements of feet and hands; sclerema of lower extremities and abdomen; cannot lift head; face normal; respiration superficial; muscles soft and flabby without apparent atrophy; knee-jerks absent; galvanic and faradic contractility almost abolished; intelligence normal.

19. Comby (*Arch. de Méd. des Enf.,* 1906, Vol. IX., p. 552).

Boy, two and one-quarter years; hands used unskillfully; can sit, but with kyphosis; can move legs, but not stand or walk; muscles soft, but not atrophic; knee-jerks absent; intellect normal.

20. Rosenberg (*Deutsch. Zeitschr. f. Nervenhk.,* 1906, Vol. XXXI., p. 130).

Boy, two and one-quarter years; family history negative; no fetal movements; had seemed entirely normal until affection of legs first noticed at eleven months, when hips and knees would give way. *Examination.*—Legs motionless except feet and toes; no atrophy; contracture at knees; knee-jerks absent; electrical contractility almost absent; great passive hypermobility; head and arms normal; face normal. Improved considerably under treatment.

21. Bing (*Med. Klinik,* 1907, Vol. III., p. 10).

Boy, two and one-half years; family history negative; dated from birth. Complete flaccid paralysis of lower extremities;

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great passive hypermobility; no visible atrophy; cannot sit or hold head erect; can make movements with upper extremities, but with diminished force; knee-jerks absent; slight diminution of electrical contractility.

22. Simonini (*Rivista di Clin. Pediat.*, 1907, Vol. V., p. 845).

Girl, ten months; family history negative; fetal movements delayed; dated from birth. Cannot sit or support head erect; diminished expansion of thorax; slight movement of arms on stimulation, none of legs; no atrophy or contracture; knee-jerks absent; strong faradic and galvanic currents produce but slight contraction.

23. Baudouin (*La Sem. Méd.*, 1907, Vol. XXVII., p. 241).

Girl, four months; family history negative; dated from birth. Entire flaccid paralysis of all four extremities, except very slight movement of fingers and toes; cannot sit or hold head erect; face normal; but little thoracic respiration; knee-jerks absent; hard, white edema, especially on lower extremities; no faradic contractility; intelligence seems normal. Died of bronchopneumonia.

24. Coombs (*British Medical Journal*, 1907, Vol. I., p. 1,414).

Girl, ten years; family history negative; dated from birth; first noticed in one hand. *Examination.*—Poorly developed upper extremities, but no apparent atrophy; grip feeble; remarkable hypermobility; same condition in trunk and lower limbs, but less marked; can walk; face not certainly affected; lordosis; knee-jerks present; no tendon reflexes in arms; diminished response to both electrical currents.

This case considered by Haberman, with some reason, a doubtful one.

25. Tobler (*Jahrb. f. Kinderhk.*, 1907, Vol. LXVI., p. 33).

Boy, one and three-quarter years; family history negative; dated from birth. Muscles well developed but soft; can make all movements of arms and legs, although very weakly; can sit, but with kyphosis, and cannot raise himself to a sitting position; cannot stand; great passive hypermobility; knee-jerks absent; faradic and galvanic contractility diminished; facial muscles rather inactive; movements of eyes normal. Decidedly improved in two years, although could not stand. Died probably of diphtheria.

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26. Lugenbühl (*Deutsch. med. Woch.*, 1907, Vol. XXXIII., p. 1,439).

Girl, five months; family history negative; dated from birth. *Examination* at four weeks.—Flaccid paralysis, with slight movements of fingers and toes; thoracic respiration almost absent; muscles of neck involved; knee-jerks absent; face normal. Improved slowly under treatment and could flex forearms.

27. Beevor (*Proceedings Royal Society of Medicine*, 1907-1908, Neurological Section, Vol. I., p. 1). (More fully reported by Collier and Wilson, q. v.)

Girl, twenty months; family history negative; apparently normal until acute illness at twelve months; after this paralysis and wasting of legs, then improved gradually. *Examination.*—All movements of arms possible but very flaccid, with passive hypermobility; condition of lower extremities similar, but much worse, and all movements weak; can sit well and support head; no local muscular wasting; knee-jerks absent; slight pin-prick awakens less notice than normal; response only to strong faradic and galvanic currents; no reaction of degeneration; intellect fair. Improved under treatment.

28. Variot and Devillers (*Bull. Soc. de Pédiat.*, 1907, Vol. IX., p. 246).

Girl, six months; family history negative; fetal movements very feeble; dated from birth. General flaccid paralysis; movements of arms and legs very feeble, but increased under stimulation; arms always kept flexed and drawn to body, and if passively extended assume original position; cannot hold head erect; kyphosis; face normal. Under massage contracture of arms disappeared. Died of bronchopneumonia.

29. Comby (second case) (*Bull. Soc. de Pédiat.*, 1907, p. 249).

Boy, four months; family history negative; general paralysis at one month after severe diarrhea, then improved rapidly except legs. *Examination.*—Legs immobile; no visible atrophy; knee-jerks absent; cannot hold head erect; kyphosis; flaccid abdominal walls with ballooning of right flank when cries; diminished faradic contractility of back of right thigh and calf, elsewhere normal; no reaction of degeneration. Improved somewhat.

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30. Leclerc (*Gas. des Hôp.*, 1907, Vol. XXX., p. 1,683). (Later report by Misserey, *Thèse de Lyon*, July 27, 1908.)

Girl, four years; family history negative; symptoms noticed at seven weeks after bronchopneumonia; then a general paralysis except face. Improved slowly. *Examination.*—All four limbs flaccid paralysis with very feeble movements; face normal; can hold head erect, but not sit unsupported; muscles soft, but no visible atrophy; hard edema of lower limbs; passive hypermobility; knee-jerks absent; electrical reactions not notably altered; no reaction of degeneration; intelligence much below normal. Improved considerably.

31. Wynter (*Proceedings Royal Society of Medicine*, 1907, Clinical Section, Vol. I., p. 48).

Fifteen months; family history negative; dated from birth. General weakness and backwardness; passive hypermobility of the joints; can sit and walk; muscles flabby but fair bulk; no reaction to faradic current; intelligence good.

Fuller report promised.

32. Ausset (*L'Echo Méd. du Nord.*, 1908, Vol. XII., p. 143).

Girl, four years; family history good; dated from birth; flaccid paralysis most marked in legs; improved to eight months, then stationary. *Examination.*—Arms move feebly, can move feet and toes, and after sensory stimulation can take a few feeble steps if supported; right leg worse; holds head erect; no passive hypermobility; knee-jerks diminished; electrical reactions normal; intellect normal.

33. Whait (*Proceedings Royal Society of Medicine*, 1907-1908, Vol. I., Neurological Section, p. 61). (Fuller report by Collier and Wilson, q. v.)

Boy, seven and one-half years; mother had weak limbs when child; several of her family could not close eyes properly; sister of patient said to have had something wrong with limbs; dated soon after birth; at three years could not close eyes completely and facial muscles weak. *Examination.*—Movements of arms and legs possible, but very feeble; muscles soft; faradic contractility diminished; contracture of hips and knees; knee-jerks absent; intellect fair. Improved slowly, and at seven years beginning to walk; knee-jerks appearing; able to use hands effectively.

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34. Thompson (*Brain,* 1908, Vol. XXXI., p. 160).

Girl, fourteen months; family history negative; dated from birth. Flaccid paralysis; cannot sit unsupported and head wobbles; arms and legs weak, but can grasp objects; some atrophy noticeable; great passive hypermobility; faradic and galvanic contractility diminished; no reaction of degeneration; knee-jerks present. Under treatment learned to sit alone, and at eighteen months was learning to stand.

35. Collier and Wilson (*Proceedings Royal Society of Medicine,* 1907-1908, Neurological Section, p. 60; *Brain,* 1908, Vol. XXXI., p. 26).

Boy, four and one-quarter years; family history good; dated from birth. Cannot sit or hold head erect; all movements of limbs possible but feeble; muscles soft and flabby; great passive hypermobility; contracture of hips and knees; knee-jerks absent; greatly diminished faradic contractility and slightly diminished galvanic; no reaction of degeneration; intelligence good. Constantly improving.

36. Collier and Wilson (second case) (*Ibid*).

Boy, five and one-quarter years; family history negative; dated from age of six months. All movements of upper extremities possible but feeble; muscles soft; great passive hypermobility; similar condition in lower extremities but less marked; contracture of hips, knees and feet; knee-jerks absent; cannot lift head from pillow; kyphosis when sitting; faradic contractility much diminished, galvanic normal. Improved, but died later.

37. Brunard (*La Clinique Bruxelles,* 1908, Vol. XXII., p. 401).

Girl, two years; family history negative; probably dated from birth. Lies helpless on back; can move legs but not stand; can grasp objects; athetoid movements; cannot sit or hold head erect; muscles soft but not atrophic; kyphosis; no passive hypermobility; knee-jerks normal; electrical reactions normal; idiot. Improved decidedly, but died of bronchopneumonia.

This case described as Oppenheim's disease, but appears to be a doubtful instance. Mental condition could possibly account for symptoms.

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38. Gastonguay (*Bull. Méd. de Quebec,* 1908, Vol. X., p. 97).

Girl, three and one-half years; family history negative; dated from birth; fetal movements not felt as in other pregnancies; condition much worse after acute illness at six weeks. *Examination.*—General flaccid paralysis, but can move arms and legs; face normal; unable to sit or hold head erect; no distinct atrophy; passive hypermobility; thickened skin on lower extremities; knee-jerks abolished; electrical reactions not tested; intellect backward. Improved in power and intellect.

39. Hartenberg (*Arch. de Neurol.,* 1909, 1-2, p. 161).

Woman, fifty years; probably dated from birth; general health good; always had feebleness of muscles, making walking difficult; grown worse for last eighteen months. *Examination.*—Weakness in grip of hands; weakness of legs; unusual passive hypermobility; kyphosis; no discoverable atrophy; no actual paralysis; electrical reaction normal; knee-jerks feeble.

This case a very doubtful one. Congenital nature is uncertain, age unusual, and electrical reactions normal.

40. Orbison (*Journal Nervous and Mental Diseases,* 1909, Vol. XXXVI., p. 204).

Girl, four years; family history negative; thought to be normal until six months, then grew irritable and less strong after exposure to heat; never sat, fed herself or talked. *Examination.*—Cannot hear or see well; internal strabismus; no optic atrophy; face normal; cannot sit or hold head erect; cannot stand or hold objects in hands; contracture of ham-strings; can move extremities fairly well, but joints seem stiff; muscles soft; faradic contractility diminished; knee-jerks obtained with some difficulty.

This case appears very atypical in the severe mental involvement and the apparent absence of flaccidity. Orbison, however, believes it a case of amyotonia.

41. Silvestri (*Gazz. degli Osp.,* 1909, Vol. XXX., p. 577).

Boy, forty-five days; brother paralytic at birth, but recovered nearly completely; maternal aunt had progressive muscular dystrophy; dates from birth. *Examination.*—Neck, trunk and limbs in complete inertia; excessive passive hypermobility; face normal; knee-jerks absent; no response to faradic current. Improved rapidly under suprarenal extract, and in four or five months could make all movements slowly.

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Case unusual in the family history, and the very rapid improvement.

42. Lereboullet and Baudouin (*Bull. et Mem. de la Soc. des Hôp.*, 1909, Vol. XXVI., p. 1162).

Boy, eleven months; family history negative; dated from birth. General flaccid paralysis; unable to sit or hold head erect; slight movements of hands and feet on stimulation; face normal; firm edema; knee-jerks diminished. Died of pneumonia.

43. Collier and Holmes (*Brain*, 1909, Vol. XXXII., p. 269).

Boy, three and one-half years; family history negative; dated from birth. General flaccid paralysis; slight movements of hands and feet; unable to sit or hold head upright; face normal; slight contracture of calf and ham-strings; no visible atrophy; passive hypermobility; knee-jerks absent; nutrition good; faradic contractility diminished.

44. Laffer (*Ohio State Medical Journal*, 1909, Vol. V., p. 609).

Girl, eight years; family history negative; dated soon after birth; first noticed in face, which was immobile when laughing or crying. *Examination.*—Lower extremities somewhat affected; can walk, but with waddling gait; upper arm muscles not strong, but good control of hands; no atrophy; shoulders, pelvic girdle and face most affected; face has mask-like expression, with drooping of eyelids; knee-jerks diminished; passive hypermobility; diminished faradic contractility.

45. Schlivek (ARCHIVES OF PEDIATRICS, 1910, Vol. XXVII., p. 34).

Girl, two years; family history good; dated from birth; slow in learning to stand or move arms. *Examination.*—Forearms extended and rotated inward; cannot flex elbows; muscles of upper and lower extremities soft and flabby; can raise arms, but chiefly from shoulders; some atrophy; passive flexion at knees limited; cannot stand or walk unsupported; knee-jerks present; intellect normal; electrical reactions show quantitative diminution but no reaction of degeneration. Improvement in the last eight months.

Somewhat doubtful case, and Schlivek himself places it in the category of muscular dystrophies. The congenital nature, however, and the fact of improvement, make it seem probably a case of amyotonia congenita.

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46. Haberman (*American Journal of Medical Sciences,* 1910, Vol. CXXXIX., p. 383).

Girl, three and one-half years; dated from birth. Can move upper extremities in all directions, but with some weakness; great hypotonia of lower extremities; muscles flabby; walks with effort if supported; knee-jerks absent; diminution of electrical contractility; intellect normal.

47. Haberman (second case) (*Ibid*). (Also described by Rothmann, *Monats. f. Psych. u. Neurol.,* 1909, Vol. XXV., p. 167.)

Girl, four months; dated from birth; fetal movements much delayed; well nourished; motionless except fingers and toes and slight movement of the head; cannot sit; knee-jerks absent; slight contracture in upper extremities; thoracic respiration diminished; quantitative but no qualitative changes in electrical reactions; needle-prick elicited only slight movement. Died.

48. Haberman (third case) (*Ibid*).

Girl, one and one-half years; family history negative; probably dated from birth, but grew worse at five to six months. General flaccid paralysis; cannot sit or hold head erect; sucking difficult; slight convergent strabismus; face normal; moves arms, fingers and feet slightly, but legs not at all; knee-jerks absent; no visible atrophy; galvanic and faradic contractility diminished, but no reaction of degeneration; intellect normal.

49. Griffith.

A number of careful studies of amyotonia congenita have appeared in addition to those mentioned. Among these may be noted those of Kundt, Rosenberg, Collier and Holmes, Simonini, Laffer, Rothmann, Misserey (*Thèse de Lyon,* July 27, 1908), Haberman, and Muggia (*Rivista di Clin. Pediat.,* 1909, Vol. VII., p. 933), and Batten (*Quarterly Journal of Medicine,* 1910, Vol. III., p. 313). I shall consequently make my analysis of the characteristics brief.

CAUSE.—*Inheritance* of any sort is almost universally absent in all the published reports. Sorgente's 2 cases were born of the same parents, but were evidently anomalous and doubtful ones. Hereditary influence seems probable, too, in the cases of Whait and Silvestri. *Sex* has no influence. *Age* is an important factor. In nearly all the patients the symptoms clearly dated from birth,

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and in most of the remainder it is very probable that they were present but overlooked. The most marked exception is the case of Beevor, in which the symptoms did not appear until the age of twelve months. In the cases, too, of Schüller and Rosenberg they were first noticed in the tenth and eleventh months respectively; and in Orbison's and one of Collier and Wilson's they seemed delayed until the sixth month. In Comby's second case symptoms were observed at one month and in Leclerc's at seven weeks. In the milder instances of the disease it may well be that only when effort to walk begins, hypotonia, already present, becomes noticeable, and it seems reasonable to assume that in all the cases there has been a congenital basis for the affection. However true this may be, it is certain that the occurrence of *some acute disease* may bring into evidence, or may accentuate, the symptoms of amyotonia. This occurred in a number of reported instances, notably those of Beevor, Comby (second case), Leclerc, and Gastonguay.

In a few instances it seems that symptoms of the disease have been *noticed before birth*, evidenced by absence or weakness of the fetal movements. We find this mentioned, for instance, in the reports of Sorgente, Cattaneo, Rosenberg, Simonini, Variot and Devillers, Gastonguay, and Haberman (second case).

SYMPTOMS.—The symptom-complex consists of a flaccid loss of power, more or less widespread throughout the body, although usually most marked in the extremities, especially the lower. The loss of power may be so great that the legs lie almost motionless and appear to be completely paralyzed. Yet, even here slight movement of the toes and fingers generally is possible, especially as a result of painful stimulation, so that absolute paralysis cannot be called a symptom of the disease. In very few instances have the children been able to stand or walk unaided; only in those of Berti (second week), Coombs, Wynter, Whait, Hartenberg and Laffer. In some of the patients the hands could grasp objects, but all movements of this sort were made, as a rule, very slowly and without vigor. In others there was decided power in the hands, but in only a few did the upper extremities appear not to be affected, viz., those of Batten (first case), Kundt, Rosenberg and Comby (second case), although in the latter they had been involved at the beginning. In the cases of Coombs and of Collier and Wilson (second case) alone was the condition worse in the upper extremities.

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There is no genuine ataxia, and although movements may be irregular and overreaching, this is dependent upon the lack of power. The lack of tone in the limbs allows of a remarkable degree of *passive hypermobility* at the articulations. This, however, is not true of every patient, nor of all the joints, even when the tone of the muscle is extremely low. Only in the severer cases are the muscles of the trunk and neck involved. When the *trunk* is affected the weakness of the muscles renders the child unable to sit erect and permits extreme functional posterior curvature when held in this position. Atony of the intercostal muscles may cause *interference with thoracic respiration.* This was noted in nine instances, viz., those of Berti (first case), Iovane, Sorgente, Cattaneo, Simonini, Baudouin, Lugenbühl, Haberman (second case) and my own. The flaccidity of the abdominal muscles may produce constipation and abdominal distention. Involvement of the *neck* permits the head to fall in any direction. In only four instances, viz., those of Batten (second case), Tobler, Whait, and Laffer, and possibly a fifth case, that of Coombs, was the *face* more or less involved. In Spiller's case swallowing, and in one of Haberman's, sucking, was interfered with, and in these and the case of Orbison's there was strabismus. The involvement of the face was decided in only 2 patients, those of Whait and Laffer. In no other instance was there any suspicion of affection of muscles supplied by the cranial nerves. Spiller's and Orbison's patients perhaps could not see well, but there is no history of optic nerve atrophy. There is no loss of control of the *sphincters* and no affection of *sensation.* The only possible exception to the latter was the second case of Haberman's and the case of Beevor's, and in neither is it very certain. It is noteworthy, however, that an unusual tolerance for strong electrical currents, so far as sensation is concerned, was observed in a number of instances. There is rarely visible *atrophy,* but the muscles are flabby and soft. It is characteristic that the general nutrition is usually good and a decided amount of fatty tissue often present beneath the skin, preventing a satisfactory palpation of the muscles. In a few instances a certain degree of hard *edema* was also noted, which added to the difficulty in observation. This was seen in the cases of Cattaneo, Baudouin, Leclerc, Gastonguay, and Lereboullet and Baudouin. The patellar, and often other, tendon *reflexes* are usually abolished or diminished. The knee-jerks appear, however, to have been little, if at all, af-

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fected in the cases of Muggia, Coombs, Thompson, Brunard, Orbison, Schlivek and Whait. In the latter they had been lost but were returning. *Contracture* has been observed in a number of cases, viz., those of Batten (second and third cases), Kundt, Rosenberg, Variot and Devillers, Whait, Collier and Wilson, Orbison, Collier and Holmes, Schlivek, Haberman (second case), and my own. Diminution of the *intelligence* is not a symptom of the disease. Occasionally, as in the cases of Beevor, Leclerc, Whait, Gastonguay, Brunard, Orbison, and in my own, more or less backwardness has possibly been noticed, and in some of those it is doubtful. In any event it appears to be an accidental complication. Only in the cases of Gastonguay, and, especially, of Brunard and Orbison, the last two rather questionable instances of the disease, was the intellect decidedly involved. The electrical reactions are characteristic, the galvanic, and especially the faradic contractility, being much diminished, even to strong currents, without any reaction of degeneration. To this there have been but few exceptions. Little or any affection of the electrical reactions was seen in the cases of Muggia, Spiller, Ausset, Brunard, and Hartenberg. The presence of reaction of degeneration would be sufficient to throw doubt on the correctness of the diagnosis.

PATHOLOGICAL ANATOMY.—A number of more or less complete pathologic studies have been made. In brief the results are as follows:—

Spiller (*University of Pennsylvania Medical Bulletin*, 1905, Vol. XVII., p. 342). No change in the nervous system; muscles pale and in some regions small, with a hyaloid appearance, indistinct longitudinal striation and preserved transverse striation; increase of connective tissue and nuclei and of fatty tissue. He regards the condition as an arrest of development. There were also sclerotic changes in the thymus gland. (Smith, *University of Pennsylvania Medical Bulletin*, October, 1905.)

Bing (*Medizin. Klin.*, 1907, Vol. III., p. 10). A portion of muscle excised during life, but nothing abnormal found except a possible increase of nuclei.

Baudouin (*La Sem. Med.*, 1907, Vol. XXVII., p. 241). Muscles pale, with increase of connective tissue and of nuclei of the sarcolemma; fatty tissue largely replacing the muscular; some of the muscle-fibers very large, others very small; disappearance of the transverse striation. Sclerosis was the predominant muscu-

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lar lesion. He regards the process as redressive. He also found arrest of development of the anterior horns of the spinal cord, with diminution in the size of the cells, some chromatolysis in the nuclei of the sixth and twelfth cranial nerves, diminution in size of the anterior spinal nerve roots, and imperfect myelin formation of the nerves. Sclerosis of the thymus and thyroid gland was also noted.

Brunard (*La Clin. Brux.*, 1908, Vol. XXII., p. 401). Doubtful case. No macroscopic lesions of nerve centers found. Histologic study promised. Nothing said about muscles.

Collier and Holmes (*Brain,* 1909, Vol. XXXII., p. 269). (Their own case.) A portion of excised muscle showed an increase of fat and of connective tissue; extreme smallness of many muscle fibers, with some increase of nuclei of the sarcolemma in places, but no degenerative changes; other fibers abnormally large, and some of these showed retrogressive changes.

Collier and Holmes (*Ibid*). (Second case of Collier and Wilson.) Muscle changes similar to those of the previous case, except for greater increase of fat and connective tissue and greater proliferation of sarcolemmal nuclei in the small fibers; fewer large fibers than in preceding case; poor development of myelin sheaths in the peripheral nerves; anterior nerve roots small and fibers small and poorly myelinated; reduction in number of cells in the anterior horns.

Lereboullet and Baudouin (*Bull. et Mem. de la Soc. Med. des Hop.*, 1909, Vol. XXVI., p. 1,162). Muscles showed some very small and other very large muscle fibers; sclerosis; nuclear proliferation and disappearance of transverse striation. The brain and spinal cord were normal.

Variot and Devillers (*Bull. Soc. de Pediat.*, 1907, Vol. IX., p. 249). Statement that autopsy was made, of which a later report is promised.

Rothmann (*Monats. f. Psychiat. u. Neurol.*, 1909, Vol. XXV., Ergnz. H., p. 162). (Autopsy on Haberman's second case.) Muscles pale and thin, with atrophy and small-celled infiltration; extensive disappearance of ganglion cells in the hypoglossal nuclei and in the anterior horns in all portions of the cord; atrophy of the anterior nerve roots.

Reyher and Helmholz (*Jahrb. f. Kinderhk.*, 1908, Vol. LXVII., p. 614). (Details given also by Haberman, l. c., p. 389.) (Autopsy report of a case of which I have been unable to find

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any clinical details published.) More or less extensive changes of the muscles, especially in the lower extremities, consisting of indistinct or absent transverse striation variation in size of the muscle fibers; replacement of portions of the muscle fibers by fat or by connective tissue with numerous nuclei.

It is evident that further anatomical studies are needed. As far as the present data will allow of conclusion regarding the nature of the disease, involvement of the muscles was present in every case studied, with the exception of that of Bing, but whether this depends upon degeneration, as Collier and Holmes believe, or upon failure of development, according to the view of Spiller, is still undetermined. As regards involvement of the nervous system, Spiller found none, Baudouin's first case showed numerous lesions, while in his second case, published with Lereboullet, none were present; Collier and Holmes found changes in the case in which a general autopsy was made; Rothmann noted extensive lesions in the cord and expressed the opinion that the majority of instances of the disease depended upon these lesions. According to Collier and Holmes the pathologic changes of amyotonia suggest those of the myopathies, but are such as admit of improvement in symptoms, and the clinical picture is not that of the myopathies. The influence of alteration of the thyroid and thymus glands has been suggested, but there is no certain proof of its existence.

It is very possible that more than one pathologic condition is included under the clinical picture of amyotonia congenita, or even, as Rothmann maintains, that transitional forms exist between this condition and instances of the early spinal muscular atrophy of Werdnig and Hoffmann.

DIAGNOSIS.—The disease is to be distinguished from poliomyelitis by the fact that the latter develops acutely in a child previously well, and that there is a degenerative reaction present. Amyotonia, on the other hand, is congenital, in the majority of cases at any rate, and probably in all. The muscular dystrophies of various types are not congenital, progressively grow worse, are localized in certain groups of muscles, and commonly show a tendency to attack several members of a family. None of this is true of amyotonia. The only instances of a familial condition in amyotonia were in the cases reported by Sorgente, Whait and Silvestri.

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Rickets may sometimes exhibit an unusual absence of power in the lower extremities, with inability to crawl, stand or walk. The association of other well-marked signs of rickets helps to distinguish it, as does the fact that such absence of power is not congenital. The pseudoparalysis of scurvy is comparatively rapid in onset and is attended by pain on movement. Idiocy of any sort is often attended by great delay in the development of muscular power, including the ability to stand or hold the head erect. In none of the cases, however, except those of Brunard and Orbison, as I have already pointed out, was there any such decided retardation of mental development.

A few words must be said regarding the unusual mobility of the joints often seen in amyotonia. The great laxness of the muscles naturally allows an increased and abnormal degree of movement. This does not appear, however, to be a necessary symptom. Unusual mobility is not referred to in a number of cases reported, and was present only to a moderate degree in my own. Moreover, I have occasionally seen great hypermobility present without any evidence of muscular atony. In fact, in one instance, the child was capable of all sorts of unusual movements and yet was remarkably robust and well developed.

PROGNOSIS.—Not sufficient is as yet known of the disease to allow of positive opinions regarding this. At least the symptoms do not grow worse, but rather slowly better, and in some of the reported cases improvement to a considerable extent has been rapid. Whether or not complete recovery can take place cannot yet be determined from the published reports. The fact that the published cases have all, with one exception, been in children would indicate either that such recovery does occur or that death takes place before adult life is reached. The death rate has been rather high. Fourteen deaths are reported, viz., Batten and Sorgente each 2 cases, Muggia, Spiller, Iovane, Baudouin, Tobler, Variot and Devillers, Collier and Wilson, Brunard, Lereboullet and Baudouin, and Haberman, each 1 case. Involvement of the thoracic muscles may, as has been claimed, be the predisposing cause of a fatal ending in some instances, as shown by the frequency of death from bronchopneumonia.

TREATMENT.—This consists chiefly in the employment of massage and electricity, and in various methods calculated to improve and sustain the general health.

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Since the above article was written the reports of the following 2 cases have come to my notice. The first case was anomalous in many respects, especially the extreme wasting with the unimpaired electrical reactions.

Ashby (ARCHIVES OF PEDIATRICS, 1910, Vol. XXVII., p. 363).

Girl, eight years; family history negative; dated from birth. In bed, almost completely paralyzed; all muscles extremely flabby and wasted, including those of the face, which has little expression: can barely move limbs; cannot sit; some difficulty in mastication; reflexes absent or feeble; electrical reactions normal; intelligence somewhat impaired. Improved slightly under treatment.

The second case seems to be a typical instance of the disease.

Moussous and Carles (*Arch. de Med. des Enf.*, 1910, Vol. XIII., p. 472. From *Jour. de Med. de Bordeaux*, 1909, No. 44, p. 697).

Girl, four and one-quarter years; family history negative; dated from birth. Cannot walk; can move arms slightly and feebly; cannot sit erect or roll over; face seems a little inert; no apparent atrophy, but muscles soft; electrical contractility lessened. No reaction of degeneration; intelligence a little diminished.

DISCUSSION.

DR. HEIMAN.—Was the Wasserman reaction taken in this particular case to rule out syphilis? After that is ruled out then it might be a degeneration of some of the nerve elements.

DR. GRIFFITH.—No, the Wasserman test was not used in this case. So far as we could learn, however, there had been at no time any symptoms of syphilis and the paralysis was in no way like syphilitic paralysis. The question of the disease being syphilitic in nature has been discussed by writers and uniformly decided against.

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CLINICAL SIGNIFICANCE OF LACK OF DEVELOPMENT OF THE PYRAMIDAL TRACTS IN EARLY INFANCY.

BY B. K. RACHFORD, M.D.,

Cincinnati, O.

It is a fact of great physiologic and pathologic importance, in the development of the spinal cord, that the fibers of the pyramidal tract are the last to become myelinated. At birth they have almost no myeline sheaths and, until their myeline sheaths are developed, it is believed that motor impulses cannot readily be carried from the brain to the spinal cord cells. It has been noted that electrical excitation of the cerebral motor cortex of dogs at birth is not followed by muscular movements (of extremities, etc.), presided over by spinal motor cells. This phenomenon was for a time explained on the supposition that the cerebral motor cortex is nonexcitable in very young animals, and this nonexcitability was thought to be due to a lack of development of cortical motor areas. While this hypothesis may serve as a partial explanation of the above phenomenon, it is now known that on or about the tenth day of the life of the dog, when the pyramidal tract has acquired its myeline sheaths, an excitation of the motor cortex will produce motion in muscles over which the spinal motor cells preside. The absence, or partial development, of the myeline sheaths of the pyramidal tract in very young animals may interfere wholly or partially with the lines of communication between the cerebral motor centers and the spinal motor cells. †"Day by day, as these myeline sheaths are developed, the cerebral and spinal motor cells are brought into closer communication, and very early in the life of the animal (ten days in the dog, and perhaps three or four months in the human infant) communication may be said to be fairly well established; prior to this time the communications are imperfect."

The above physiologic facts are of great pathologic importance, and if we had sufficient data to rightly interpret them in their clinical bearings on the neurotic disorders of infancy they would throw much light on some of the unexplained clinical manifestations of these diseases.

† "Neurotic Disorders of Childhood." Rachford, 1905.

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I hope I may be indulged in some suggestions and reflections as to the possible rôle which this lack of development of the pyramidal tract may play in producing nervous symptoms in early infancy.

A—Convulsions in Infancy.

The above physiologic facts may be offered in explanation of the comparative immunity which young infants enjoy from convulsive disorders during the first few months of their lives. It is probably true that during the first months of life the motor areas of the cerebral cortex are not sufficiently well developed to readily respond to stimulation, and it is also true that at birth the peripheral endings of sensory nerves are not so perfectly developed or so keenly sensitive to reflex and other stimuli as they are some months later, and it is also true that during this period of life the nervous system of the infant is not so much exposed to irritation by systemic and gastrointestinal bacterial toxins as it will be a few months later. It is also possible that, while all of these causes may be operative in protecting the young nervous system from convulsive disorders, the most potent factor operating toward this end may be the lack of development of the pyramidal tract. Such, in fact, is my belief. Irritation of the motor cortical centers and of the basal convulsive centers may produce a discharge of nerve force from these cells, but the motor impulse, through lack of development of the pyramidal tract, is imperfectly, or not at all, communicated to the spinal motor cells, and, therefore, no convulsion results.

But even in these early days of life it is not probable that the paths of communication are so completely closed that very powerful stimuli may not send an impulse from the brain to the cord. As the infant grows older, however, the communications between the brain and cord, through the pyramidal tract, become better and better established, and, as a result, convulsive disorders become more and more frequent as these communications are perfected. It is probable that the period when the pyramidal tract reaches full development may vary greatly in different children.

B—The Plantar Reflex and Babinski's Sign.

In 1898 Babinski called attention to certain variations in the plantar reflex produced by disease of the pyramidal tract. He observed that in cases presenting a lesion which interfered with the conducting power of the pyramidal tract, there were present

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certain variations from the normal plantar reflex. In normal adult individuals the reflex, elicited by plantar irritation, is *flexion of the toes;* in pyramidal disease, however, the response to this reflex is *extension of the toes.* This observation of Babinski has been confirmed by many observers, so that at the present time extension of the toes (especially the big toe) is recognized in the adult and older child as a valuable sign of pyramidal disease, and the accepted physiologic explanation of this sign is that it is brought about by the more or less complete destruction of the conducting power of the pyramidal tract, thus interfering with the lines of motor communication between the brain and the cord. In this manner certain inhibitory influences, which the brain exercises over spinal reflex movements, are wholly or partially cut off.

Babinski also observed that this extension of toes, which is the pathologic reflex response to plantar irritation in the adult and older child, and which is due to the severing of the pyramidal lines of communication between the brain and the cord, is the normal reflex response in young infants. In infants under one year of age *extension of the toes* is the normal response to plantar irritation, because of the incomplete development of the pyramidal tract at this age. This explanation was offered by Baginski and has been accepted by a number of recent observers.

That the normal *toe-extension* reflex of infancy is less constant and less deliberate than the pathologic toe-extension reflex of the adult does not militate against the above explanation. The unreliability and inconstancy of the toe-extension reflex in infancy (as pointed out by Morse and others) may be explained by the incomplete and irregular development of the reflex spinal centers in the young infant and by the variations which individual infants present in the development of their pyramidal tract, but the fact remains that toe-extension is the normal plantar reflex in young infants, and lack of development of pyramidal tracts is the only rational explanation of this phenomenon.

C—Infantile Cerebral Palsies.

It is a well recognized fact that in the cerebral palsies of early infancy, produced by cortical hemorrhages and other lesions affecting the motor centers of the young infant, the lesion may precede by many months the appearance of the spastic paralysis which is characteristic of this condition. This is especially true

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of the cerebral palsies produced by natal and prenatal injuries to the brain. In speaking of these cases, Sachs* says: "It is a matter of fact that the symptoms of such intrauterine defects are not always manifest at birth, and, indeed, a number of months may pass before it becomes evident to the physician that the child's cerebral condition is not a normal one. A very fair percentage of cases which appear to begin during the first years of life could properly enough be classified among the prenatal palsies, and if in any child the first symptoms of a cerebral palsy are noticed several months after birth, and yet the period of labor was entirely normal, I should be inclined to classify that case rather among the prenatal palsies than among the birth palsies."

While it is a well known fact that injury to the cortical motor areas of an infant before or during birth may not for *some months* be followed by the characteristic spastic palsies of the extremities, yet the explanation for the delay in these symptoms is not apparent. The question, therefore, arises: May not these clinical phenomena be explained by the late development of the pyramidal tract? At birth the fibers of the pyramidal tract, by reason of the absence of myeline sheaths, afford but very imperfect lines of communication between the cerebral motor cortex and the motor cells of the cord. An injury to the cortex, therefore, may not be communicated to, and may not influence the functions of, these cells, and, as a result, the functions of the spinal cord in the infant are, for the time being, not disturbed. But as the days and months pass certain of these pyramidal fibers not directly involved in the lesion, take on their myeline sheaths, and along these lines the injured region in the cortex is gradually put in closer communication with the spinal motor cells, and as a result the spastic paralysis gradually develops, producing the characteristic clinical picture about the fourth month.

* "Nervous Diseases of Children." Sachs.

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GENERAL CONSIDERATIONS REGARDING THE EFFECTS OF VACCINES.

BY S. McC. HAMILL, M.D.,
Philadelphia, Pa.

When an individual becomes the victim of a bacterial infection the protective powers of the body are immediately called into action. If these protective forces play their part effectually an indefinitely enduring immunity is eventually established.

Many theories have been advanced in explanation of this process. To this the comparatively recent discoveries of Wright and his co-workers have contributed not a little. They have shown that there is present in normal blood a fixed amount of a protective substance which they call opsonin, and which has as its function the preparation of bacteria for ingestion by leukocytes, which is specific in its action. Infected individuals have less of this specific opsonin than do normal persons, but by injecting a vaccine prepared from killed cultures of the infecting organism, the amount of opsonin may be increased, and by repeating such injections at properly timed intervals, it may be maintained at or above the normal level and, by thus rendering the blood rich in protective substances, hasten the establishment of immunity.

The method which Wright determined for measuring the opsonic power of the blood is as follows: Equal parts of washed corpuscles, an emulsion of the microorganisms and serum from the patient whose blood is being tested are mixed in a capillary pipette and incubated at 37° C. for fifteen minutes. From this mixture a film is prepared, and the number of organisms engulfed by, say fifty polymorphonuclear leukocytes, are then counted under the microscope and compared with unity—which is the number found in fifty polymorphonuclear leukocytes in a similarly prepared film made from a mixture of equal parts of the same washed corpuscles, the same emulsion of bacteria, and the serum of a normal individual. The resulting factor he terms the opsonic index. Immediately following the injection of killed organisms there occurs a fall in the opsonic content of the blood, the so-called negative phase. This fall is followed, after a period varying from a few hours to perhaps several days, by a rise in the index to or above

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the normal. Wright and many of his followers have insisted upon the frequent determination of the index as a guide to treatment, because they believe that the repetition of injections during the period of lowered index may prolong it, and by thus reducing the resistance of the patient contribute to the advance of the infection.

There has been much controversy over this particular point, those in opposition to Wright—who now compose the majority—holding that the danger of the occurrence of the so-called cumulative negative phase has been exaggerated, and that a careful study of clinical symptoms is quite sufficient indication for the timing of the dose. Without question, experience has demonstrated the correctness of this latter view—a very much to be desired result, as the elaborate and delicate technique involved in the establishment of the index would have interfered materially with the extensive application of a therapeutic measure which gives promise of valuable results.

The method of preparing the vaccines is comparatively simple. The organism to be used is grown in pure culture upon a satisfactory medium. This culture is then taken up in normal salt solution, which is shaken until it is evenly distributed, after which it is standardized so that each c.c. of the emulsion contains a known number of bacteria. It is then heated to 60° C. for thirty minutes to kill the bacteria, and 0.5 per cent. of carbolic acid is added as a preservative. The total number of bacteria per c.c. being known, the requisite number for the individual dose can be obtained by using a corresponding amount of the emulsion.

The application of vaccine therapy to the treatment of disease in children differs in no respect from its application to the treatment of disease in adults, save as to the dosage, and this, as in the administration of all other remedies, is based upon the age. The dose varies, however, for both adults and children according to the organism injected—thus the number of dead staphylococci that may be introduced into the body with perfect safety is many times greater than the number of dead streptococci. For the individual organism, also, the dose varies within rather wide limits. The majority of investigators advocate relatively small beginning doses, but they vary greatly in their opinions as to the maximum limit. The question is fortunately of no serious importance, as disturbing results have rarely followed the use of the larger doses.

The reports referring exclusively to the vaccine treatment of diseases in children are few in number. Children are included oc-

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casionally in papers dealing with the treatment of both adults and children, but in these it is usually impossible to measure the effects of the treatment as applied to children because they have not been considered separately in the general result.

Of the gonococcal infections of early life, vulvovaginitis is by far the most common. The ophthalmias of the newborn are more serious, but unfortunately they do not offer a very promising field for vaccine therapy, on account of the acuteness of their course. No communications regarding the vaccine treatment of such cases have been noted, but there are a few, chiefly unfavorable, reports, regarding the treatment of gonorrheal infections of the eye in adult life. A few isolated cases of gonococcus infection of the joints in children have been treated by vaccines, but this treatment has been much more widely applied in adults.

Cole and Meakins, White and Eyre, Irons, Eyre and Hartwell collectively report 124 adult cases. They all report favorably upon the treatment and some of them are enthusiastic.

Alice Hamilton and Jean M. Cooke (*Journal of Infants' Diseases,* 1908, Vol. V., p. 158) were the first to apply bacterial vaccines to the treatment of *vulvovaginitis.* They studied the effect of gonococcal vaccines in the treatment of 67 cases, and their conclusions in part were as follows:—

"It is sometimes impossible to raise the opsonic index to the gonococcus by injections of dead gonococci, but when this occurs an improvement in the clinical condition usually follows.

"Acute cases inoculated with killed gonococci from strains which have been grown for several months on artificial media improve rather more rapidly than do control cases which receive no injections of killed gonococci.

"Better results are obtained by the use of strains which have been grown for a long period of time on artificial media than by the use of freshly isolated strains, and there appears to be no advantage in using the patient's own organism.

"While inoculation treatment does not produce a marked effect during the first weeks in acute cases, it seems to shorten the later stages; in chronic cases its effect is more evident than in acute.

"The substitution of inoculation therapy for local treatment in little girls presents some decided advantages, especially in chronic cases."

These conclusions are fully justified by the detailed results described in the body of the paper.

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Butler and Long (*Journal of American Medical Association,* 1908, Vol. I., p. 744, and *Ibid.,* Vol. II., p. 1,301) have contributed two articles on this subject, in which they report 37 cases. In their first series they obtained more satisfactory results in the protracted cases from the vaccine made from four strains; but in their second series they concluded that such vaccine had no advantage over one prepared from a single strain. They believe that gonococcus vaccine is more effective than local treatment, and that in some cases it produces a very rapid improvement and often recovery, evidently using the term "recovery" in a relative sense.

Churchill and Soper (*Journal of American Medical Association,* 1908, Vol. II., p. 1,298) have published the results of the treatment of 41 cases, but 18 of which they were able to follow continuously. Of 9 cases which they could trace after treament, 6 continued free from all evidence of the disease for periods ranging from three weeks to five and a half months. They used old cultures from one strain, and, like Hamilton and Cooke, believe them preferable to fresh strains. They conclude that the inoculation treatment seems to shorten the stay of patients in the hospital, but they did not determine that it actually lessened the total duration of the disease.

By far the most favorable results have been published recently by Hamilton (*Journal of American Medical Association,* 1910, Vol. I., p. 1,196), and for this reason his results are given in greater detail. He reports 344 cases of vulvovaginitis of gonococcal origin, 84 of which were given vaccine treatment. The author used three vaccines—one was prepared from a sixteen to eighteen-hour agar culture from acute urethritis in the male, prepared after Wright's method in the Rockefeller Institute, the strength of the emulsion being 100,000,000 to 1 c.c.—the second, a vaccine from stock culture of the same strength, prepared at the Presbyterian Hospital—and the third, a vaccine prepared by Parke, Davis & Co. No homologous vaccines were used. The smallest number of injections to produce a cure was four, and the greatest eighteen. He began treatment in all cases, except those under six months of age, with 50,000,000 every fifth day, increasing the dose by 10,000,000 until five injections were given; following this the intervals were ten days. In most of the acute cases, six injections were sufficient for a complete cure. The cure consisted of an absence of gonococci after four bacterial examinations of the secretion from the vagina, made at intervals of a week, followed by two examina-

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tions at an interval of two weeks. Nineteen children returned to the clinic after a period of three months without evidence of the disease.

Of the 84 cases, 16 were of long standing. The more chronic the case the larger was the dose required. Of these 16 chronic cases which had been under irrigation treatment for long periods, 3 were not benefited, 1 did not return and 12 were cured. Those which did not respond to treatment with one of the vaccines frequently did well on the others. When no benefit was noticed, a new strain was used.

He obtained three cures by vaccines in children under one year of age, the youngest being three weeks old. Of the 260 cases treated without vaccines by irrigation, the average length of the treatment was ten months; while the average length of treatment in the 84 vaccine cases was 1.7 months. No local treatment was used in any of his vaccine cases.

It is extremely difficult to explain the difference in results reported in this paper and the ones which precede it. Whether or not the fact that Hamilton changed from one vaccine to another, when he found that his results were unsatisfactory with some particular strain, is sufficient to explain the discrepancy cannot be determined, because he does not indicate the frequency with which this procedure was resorted to. His cures were not only frequent, but generally permanent, and often after a comparatively short course of treatment. It is greatly to be desired that his results may be confirmed in the hands of other investigators.

Scarlet Fever.—The subject of the prevention of scarlet fever by injections of streptococcus vaccines seems to have been very widely studied in Russia and scarcely at all elsewhere. A very excellent review of this work has been published in the *Boston Medical and Surgical Journal* (1910, pp. 162, 242) for February of the present year, in an article by R. M. Smith, from which the following is largely quoted:—

This treatment had its origin some years prior to the studies of Wright and his school, and was inaugurated by Stickler, who, in 1883, injected into himself the blood of a scarlet fever patient. The result was a scarlatinal eruption on the third day, which was followed by desquamation. He later injected the desquamated scales into some altruistically inclined individuals, producing only a localized hyperemia, but apparently establishing an immunity, as

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proven by failure to produce a recurrence of the symptoms by re-injecting these subjects at later periods.

The treatment was not considered again until 1905, when Gabritchewsky (*Russ. Vratch.*, 1905, p. 80, and *Ibid*, 1906, pp. 10, 469) used a streptococcus vaccine prepared from boullion cultures of a streptococcus isolated from a person ill with scarlet fever.

The reaction which follows this treatment is either an area of redness and inflammation around the site of the inoculation, which is somewhat painful and tender, or in from 10 per cent. to 15 per cent. a punctate erythema, very much like that of scarlet fever, but which is not followed by desquamation. The only reaction to the first injection is a slight elevation of temperature. The second injection may be followed by sore throat or swelling of the glands and a strawberry tongue, and in very rare instances the reaction may be sharp enough to produce albuminuria and prostration. Less often there is no reaction after the second injection and practically none after the third. A few cases give no reaction, especially those which have had scarlet fever. Only one fatal case in 50,000 injected had been recorded; this case having had a severe nephritis at the time of the injection.

Smith gives statistics from the articles of seventeen Russian writers, covering 4,672 individuals, 40 of whom later developed the disease. Of this number, 127 were inoculated by Gabritchewsky. Of his cases, 5 developed the disease, 4 of these after the first treatment. Of 90 children not vaccinated and living in the same district, subject to the same conditions, 36 developed scarlet fever.

In the experience of all the writers, a vast majority of the cases occurring after vaccination developed the disease before the second vaccination could be given. The results seem quite remarkable, but the method has probably failed of popularity outside of Russia, because of the severity of the symptoms of reaction.

Vladimiroff (*Arch. f. Kinderheilk.*, 1909, Vol. LII., H. 3, p. 28) gives a brief review of the Russian literature relating to this subject and states that 50,000 vaccinations had been made, and that the procedure is harmless. He reports 1 case of his own, in which he thought immunity had been established.

There are a few scattered reports of the treatment of isolated cases of scarlet fever by bacterial vaccines, which have been in the main unsatisfactory, but there have been some apparently favorable results in the treatment of the complicating ear and

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glandular suppurations by vaccines prepared from streptococci isolated from cases of scarlet fever.

The majority of investigators have considered the acute generalized infections as not well adapted to treatment by bacterial vaccines. Nevertheless, one finds very brilliant results reported in the treatment of such cases.

It is very difficult to interpret the results of treatment in any acute self-limited disease, and there is probably none of which this may be stated more truly than *pneumonia,* especially the lobar pneumonias of early life.

There have been comparatively few studies of the effect of vaccine treatment upon pneumonias in children. In a paper by Hoobler, to be referred to later, 1 case was included, and one finds children included here and there in general reports upon pneumonia. Thus Wilcox and Morgan (*British Medical Journal,* 1909, Vol. II., p. 1,050) treated 24 cases of lobar and primary bronchopneumonia due to the pneumococcus, using autogenous vaccines in all cases. Fourteen of their 24 cases were children ranging in age from two and one-half to fourteen years. Twenty-two of the 24 cases treated recovered. They thought the duration of the disease was shortened in some cases. The most striking effects, however, were shown in the cases which had been running protracted courses, and in these they considered the vaccines responsible for the cure.

Harris (*British Medical Journal,* 1909, Vol. I., p. 1,580) includes two children in reporting a series of 11 cases. One of these had an unresolved pneumonia of some weeks' duration and the other a complicating empyema and nephritis. In the latter cases the vaccines had a remarkable effect upon the complications, and both cases recovered.

Wolf (*Journal of Infants' Diseases,* 1906, Vol. III., p. 731) concludes from the treatment of 14 adult cases that the vaccine treatment of patients with lobar pneumonia seems to exert a favorable influence upon the course of the disease and to hasten the crisis.

Leary (*Boston Medical and Surgical Journal,* November 11, 1909) noted rapid relief of toxic symptoms, especially the quick cessation of delirium. He treated 83 cases, with a mortality of 9.7 per cent., and 15 of these cases had crises within three days.

The most striking feature in these reports seems to be the early crisis in the cases which recovered. Practically all of the authors

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had distinctly lower mortalities in the vaccine-treated cases than occurred in the others they were observing in the same seasonal periods, and their cases were not supposed to have been selected.

Staphylococcus Infections.—Treatment by staphylococcus vaccines, especially in such localized infections as furunculosis, carbuncles, abscesses and acne, has been very effective. Indeed, all forms of infection due to the staphylococcus pyogenes aureus seem to offer a promising field for treatment by vaccines. A few groups of cases of furunculosis in children have been reported, and one finds scattered cases of other forms of staphylococcic infection in early life which have responded satisfactorily to vaccines.

Thus several instances of Ludwig's angina (Wright, Walters, Coombe and Solly) have been successfully treated, and Mallanah (*British Medical Journal,* 1909, No. 2, p. 984) reports a very interesting case of cancrum oris, which was failing rapidly, in spite of local treatment, and which was thought to have been in a hopeless condition when vaccine treatment was begun. Three injections at intervals of three days resulted in complete cure at the end of two weeks.

Wechsel and Michaelis (*Deutch. Med. Woch.,* July 29, 1909) treated 13 children with staphylococcic abscesses with very satisfactory results. Other forms of treatment had been tried without effect. Two of their cases were infants, aged four months. In addition to having abscesses, they were both syphilitic and in a wretched state of nutrition. The treatment not only cured their abscesses, but improved their nutrition in a striking manner—a result which has been noted by other observers.

There are many infections in which the staphylococcus acts as a secondary factor, and in these the addition of a staphylococcus vaccine to the treatment will frequently result in a prompt disappearance of disturbing symptoms.

Autogenous vaccines do not seem as essential in the treatment of infections with the staphylococcus as in infections with some other organisms, but they are always preferable, and there are a good many instances on record in which improvement has followed the use of vaccines made from the patient's organism after stock vaccines had failed.

Treatment by *streptococcus* vaccines has been unsatisfactory. The results with stock vaccines have been especially bad. The great variety of different strains of the streptococcus, the presence of several strains in the same infection—some virulent and

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some avirulent—have not only made treatment by stock vaccines unsatisfactory, but have also rendered uncertain the results from autogenous vaccines. They have been used almost entirely in adults.

Colon infections of the urinary tract are being treated very extensively by colon vaccines at the present time; much more, in fact, than the contributions to the literature would indicate. Their use has been restricted almost exclusively to adults. They seem to give very prompt relief to the symptoms, but to have no effect upon the disappearance of the organisms from the urine.

Otitis Media.—Vaccines have been used to some extent in infections of the middle ear. Tunicliffe obtained benefit in some cases which were due to the pseudo-diphtheria bacillus, the cases having occurred subsequent to attacks of scarlet fever.

A case of infection with the bacillus pyocyaneus of more than two years' standing reported by Mallanah (*British Medical Journal,* 1909, No. 2, p. 934) was completely cured by six injections of an homologous vaccine. Every other form of treatment had been tried without success.

Trimble (*Journal Kansas Medical Society,* 1909) reports 4 cases; the organisms being respectively the staphylococcus aureus in 2 cases; the bacillus pyocyaneus in 1; and the bacillus pyocyaneus with the staphylococcus albus in 1. In the last case he used a mixed vaccine, and recovery ensued in all. He advises that in all cases of chronic otitis media in which the symptoms are scarcely sufficient to justify operative procedures, vaccine treatment should be employed.

A very interesting paper recently published by Hoobler (ARCHIVES OF PEDIATRICS, Vol. XXVI., p. 674, 1909) reports a series of 8 cases, including one each of the following conditions:—chronic bronchitis, following whooping cough, complicated by acute purulent otitis media due to the staphylococcus aureus; bronchopneumonia with delayed resolution due to a mixed infection with the staphylococcus aureus and a streptococcus; a general rheumatic infection, due to the staphylococcus aureus; a septic infection after operation for double mastoid disease due to the staphylococcus aureus; a streptococcic empyema following pneumonia, a streptococcic septicemia following acute purulent otitis media; a staphylococcus furunculosis; and a streptococcus forunculosis following infection from hypodermic injections. Practically all of these cases were treated by autogenous vaccines and

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the mixed infections by mixed vaccines. Recovery ensued in all, and in several the cures were unusually rapid. Hoobler was of the opinion that the recovery in the cases of general rheumatic infection and streptococcic septicemia could be justly attributed to the vaccines, and that measured from the standard employed in judging of the efficiency of drugs, the vaccine showed a favorable influence in all the cases.

Floyd and Worthing (*Boston Medical and Surgical Journal*, 1908, No. 5, p. 158), in a general paper, report 9 cases in children, including 2 of empyema, 1 each of osteomyelitis, abscess of staphylococcus origin, generalized furunculosis, slowly resolving pneumonia complicated by empyema, pyelitis due to the bacillus Escherich, of the colon group, pyelitis due to another variety of the colon group, and an abscess of the right thigh due to the pneumococcus. The case of osteomyelitis, which terminated fatally, was a systemic infection with the staphylococcus aureus. The two empyema cases were pneumococcic infections and both responded promptly to drainage and injections with stock vaccines. All of the remaining cases ended favorably.

White and Eyre (*Lancet*, 1909, No. 1, p. 1,586) include in a report of a number of adult cases, one of a boy aged twelve years with ulcerative colitis. The condition had persisted for four years. Injections with vaccines prepared from a bacillus of the colon group isolated from the feces and given in doses of from 5,000,000 to 50,000,000 at intervals of one week resulted in a complete cure at the end of three months.

Tuberculosis.—The opinions regarding the dose of tuberculin for the treatment of tuberculosis in early life are largely in favor of the smaller doses. For children under one year the dose has varied, almost without exception, from 1/12,000 to 1/8,000 mgs., and for children from one to twelve years of age from 1/4,000 to 1/1,000 mgs. On the other hand, Schlossman (*Deutsch. med. Woch.*, February 18, 1909, p. 289) strongly advocates, what would seem in the light of more recent discoveries, the use of remarkably high doses. He says, regarding the dosage, that:—

(1) It should be so gauged as to produce as little harm as possible.

(2) It should be as large as possible, provided no harm is done the patient.

In relation to this second statement, he points out that once toleration has been established for a small dose the body of the

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tuberculous subject is much more readily rendered immune to large doses. He, therefore, begins his treatment with relatively small doses; for instance, in 2 infants, whose cases are described in detail, he begins the treatment in one with 1/100 of a mg., and in the other, aged eighteen months, with 1/10 of a mg. In support of the larger doses, he states that tuberculous antibodies are not produced in the child until doses of about 1/10 gm. have been reached, and that the quantity of antibodies formed increases parallel with, or in proportion to, the quantity of tuberculin injected. So long as the antibody content is maintained, further injection of tuberculin is not followed by reaction, hence the antibody content in the serum is of some value as an indication for pursuing or interrupting the injections. The index should be maintained at a certain height throughout the treatment.

In the light of his belief that antibodies cannot be demonstrated until a dose of about 1/10 gm. has been reached, he aims to bring these patients to the point of tolerating doses of decigrams and grams, since this assures the antibody formation a phenomenon which is associated closely with the process of recovery.

He divides the tuberculin treatment into two stages:—

(1) That in which the organism must be brought to a point of tolerating the amount of tuberculin necessary for the production of antibodies.

(2) The maintenance of antibody formation by the use of sufficiently large quantities of tuberculin at suitable intervals.

For children on prolonged treatment he gives tuberculin in doses of from .5 to 2 gms. repeated from every five to eight days. He insists upon it that immunity to tuberculosis must not at any time be allowed to diminish, so that the patient cannot tolerate .1 gm. practically without reaction.

Schlossman acknowledges that bad effects occasionally occur, and the most important one is the failure of a reactive temperature to return to normal. In these cases the infant sometimes succumbs and miliary tuberculosis is found at the autopsy. He believes, however, that such accidents can be avoided. Children liable to such occurrences always show a great tendency to irregularity of temperature even before the injection.

He points out that cases of this character are practically certain to succumb to the disease anyhow, and he thinks that these cases should be treated, but that the utmost care should be taken

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to use small initial doses. Once the organism has become accustomed to the minute initial dose and the resisting forces called out by the injection of large doses, the danger of miliary tuberculosis may be set aside. As a further support of the larger doses, Schlossman contributes some rather striking results. For instance, he reports the case of an infant in whom treatment was begun at the age of nine months for tuberculosis of the bronchial glands and lungs. The child was living and in good health at the age of one and three-quarter years. In this case the child reacted twenty-eight times in the course of sixty days on a dosage of 1/10 of a mg., finally the organism became habituated to this dose of tuberculin and no reaction occurred thereafter.

Eight other tuberculous infants systematically treated with tuberculin survived the first year of life. On the other hand, 4 not treated with tuberculin died.

Regarding the prognosis of tuberculosis in infants, he makes the following statement: "I regard it in the main as very unfavorable, but if a child is suitably fed and scientifically treated with tuberculin, the prognosis may be regarded as favorable in the sense that such infants, if they are in good condition, may be carried through the difficult period of infancy." He does not intend to imply that a complete cure is established, but he does believe that healing to some degree takes place in the glands or in the lung tissue, thus preventing miliary diffusion, which is the common termination of untreated cases.

Engel (Brauer's *Beitr. z. Kl., d. Tub.*, 1909, No. 13, p. 245), in reporting upon the use of tuberculin in the treatment of tuberculosis in children, follows very much the same line of dosage as that administered by Schlossman, except that his beginning doses were smaller. He gradually increases the amount of tuberculin to the point at which a febrile reaction occurs. He then continues this dose until no febrile reaction takes place, and then again advances the dose to the point of tolerance until as much as 2 mgs. are given. He does not fear febrile reactions, but believes that within limited degrees they are advantageous.

In the treatment of children suffering from benign forms, which he defines as the glandular and osseous, he considers it perfectly safe to give 1 mg. as the initial dose. With two or three such doses he is usually able to establish immunity.

In pulmonary tuberculosis, in which the lesion is not severe, one can usually begin with 1/10 gm., but in the severe cases

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the beginning doses should be from 1/100 to 1/1,000 mgs. He states that in determining the maximum dose he found it very useful to determine the antibody in the serum. He considers the most important thing in treatment as follows: (1) To establish a high degree of immunity, and (2) to maintain it for as long a period as possible.

The antibodies of the serum may be determined as an indication of the degree of the immunity; but he believes that the body temperature may be safely adopted as the sole guide in treatment.

The intervals between the doses should not be more than four or five days. This interval is chosen because it approximately measures the duration of the complete immunity. The time required for producing immunity to large doses of tuberculin is from two to three months.

In endeavoring to reach some conclusion as to the results of tuberculin treatment, he states that only years of observation will enable one to determine definitely that a cure has been effected. He has been enabled, in many instances, to clearly demonstrate a lessening of the anatomic lesion, but at the autopsy in some fatal cases he observed an extensive fibrous formation in the cadavers of children with tuberculin at an age when there is no spontaneous tendency to anatomic methods of healing. These manifestations he accredited to the tuberculin. He points out that it has already been demonstrated by pediatrists that an infant which has been infected with tuberculosis cannot survive longer than a year. He refers to eight instances, between the ages of three and eight months, having various forms of tuberculosis, chiefly glandular, none of them occult, which have successfully passed through the period of early infancy.

He concludes that tuberculin exercises a favorable influence upon the disease, and that it may be positively asserted that it tends to impede the progress of the disease, and that it can be given without in any way injuring the child's development.

Apropos of the method of healing as defined by Engel, it may not be amiss to here refer to the statement of Ochsner (*Southern Medical Journal*, 1909, No. 2, p. 445) regarding the vaccine treatment of joint tuberculosis that "vaccine treatment, added to his former routine method, has resulted in recovery with greater mobility of the joints." He explains this as being due to a vascularization of the part—a tearing down of the connective tissue wall, thus giving the phagocytes an opportunity to destroy

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the tubercle bacilli, a process which is quite contrary to the old accepted theory of cell proliferation, connective tissue formation, constriction and elimination of blood vessels resulting in fatty degeneration, necrosis, and finally calcareous deposits.

He reached this conclusion in the following manner: In treating some cases of tuberculous cervical adenitis, he operated upon one side of the neck, and then began the administration of vaccines. Some time later he operated upon the other side. At the first operation the glands had their ordinary gland capsule, and, in addition, a very considerable deposit of periglandular connective tissue. At the second operation, on the opposite side, the gland capsule was about the same, but the periglandular connective tissue had almost entirely disappeared, and the surrounding tissues were much more rich in blood vessels. Both these views—that of Engel and that of Ochsner—are based upon pathologic evidence, but are diametrically opposed. The older pathology corroborates the view of Engel, whilst the theoretical viewpoint of the vaccine therapist based upon his clinical experiences, suggests that in lesions of any sort, and especially tuberculous lesions, walled off by fibrous tissue, satisfactory results from bacterial vaccines cannot be obtained until in some manner the vascularity of the localized lesion is increased.

To turn from favorable results of treatment with these relatively large doses to a consideration of treatment by doses varying from 1/10,000 to 1/1,000 mgs., which also show favorable results, rather confuses one's judgment as to the proper method of treatment to follow. The advocates of the smaller doses are very definite in their statements regarding the danger of larger doses. Thus Dingwall Fordyce (*Lancet*, 1909, No. 2, p. 1,745), in reporting upon the treatment of 100 children, lays great stress upon the danger of large doses, or the use of tuberculin at all in cases where the temperature is unsteady. His results in the treatment with tuberculin, however, have been in no sense as satisfactory as Schlossman's; and he is rather inclined to believe that treatment by rest, climate, feeding and careful nursing yields as good results as tuberculin treatment. If these measures cannot be adopted he thinks that tuberculin can be used to great advantage.

Reviere (*British Medical Journal*, 1907, No. 1, p. 859) reports very excellent results by treatment with doses varying from 1/12,000 to 1/3,000 mgs. This report consists of 16 cases ranging in age from one to twelve years, and including cases of

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dactylitis, superficial abscess, tuberculous glands, psoas abscess, tuberculous joint disease, abdominal tuberculosis and phthisis. He lays great stress upon the point that his results have been more satisfactory with small doses at short intervals than with larger doses at longer intervals.

Carmalt-Jones (*British Medical Journal,* 1908, Vol. II., p. 581) reports a series of 367 out-patient cases treated with tuberculin in St. Mary's Hospital. Of this number, 155 involved lymphatic glands; 87 of these he was able to follow after treatment; in 8 of this group the injection had been employed as an adjunct to removal by operation. All were cured. Of the remaining 79, 27 were cured, 22 much improved, 18 improved, 8 unchanged and 4 worse. Twenty-four of the cases were in children under ten years of age. His results were best in this group, and increasingly worse with advancing age, except for the ten years following puberty. In the children treated, he gave as the maximum dose for those under five years of age 1/10,000 mgs. and for the older children 1/4,000. Whilst he obtained fairly favorable results in the many other tuberculous conditions treated, his most favorable ones were in lymphatic tuberculosis.

Carmalt-Jones's experience has been the common one of those who have used tuberculin extensively in the treatment of tuberculosis. Favorable results have been obtained by various authors in the treatment of ulcers, sinuses, abscesses, lupus, dactylitis, bone disease, ocular tuberculosis, peritonitis, tuberculosis of the genitourinary tract and pulmonary tuberculosis.

Strikingly good results were obtained by Smith (*British Medical Journal,* 1909, Vol. II., p. 1,046) in the treatment of tuberculosis arthritis. In this group there were 17 children of thirteen years and under, and of this number, 11 were cured, 4 improved and 1 unimproved. Both he and Reviere lay great stress upon the importance of treating associated infections which they found in several instances to be due to the staphylococcus. Such cases frequently recover promptly when the treatment of the added infection is begun.

In summing up, it may be repeated that the results from the vaccine treatment are much more satisfactory in early life than in later life, and localized tuberculous lesions, especially the glandular types of tuberculosis, are much more favorably affected than the pulmonary type, especially those with generalized symptoms.

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There is a long list of additional infections, such as erysipelas, diphtheria, cerebrospinal meningitis, pertussis, dysentery, infective endocarditis, pyorrhea alveolaris, actinomycosis, infection of the sinuses and various infections of the skin which have been treated by vaccines, but the number of cases included in these reports have been so few or the results so indefinite that it has seemed unnecessary to do more than refer to them. The results in diphtheria and cerebrospinal meningitis have been so inferior to the results from serum treatment that the use of vaccines in these conditions seems scarcely justifiable.

The impressions which one gathers from a thorough study of the literature of vaccine therapy as it relates to both adults and children are that it has been clearly demonstrated that localized infections with the staphylococcus pyogenes aureus are quickly cured, especially when autogenous vaccines are used, that generalized infections with this organism are favorably influenced, that gonococcal joint infections, which are relatively rare in children, are usually benefited, and as to other forms of infection that there are not enough data at hand to justify one in drawing conclusions as to the value of bacterial vaccines in their treatment.

It is true that in individual and even in small groups of cases, striking results are reported which seem attributable to the vaccine treatment, but one must not forget in measuring the value of any treatment that striking recoveries sometimes occur in seemingly hopeless cases, irrespective of the nature of the treatment.

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THE USE OF BACTERIAL VACCINES IN CHILDREN'S DISEASES.

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AND

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At the outset it should be said that the credit for the technical part of the work belongs entirely to Dr. Hoobler. He made the cultures, prepared the vaccines and administered them. The cases upon which they were used occurred in the service of the children's wards at Bellevue during the past ten months. We observed them together and the conclusions reached are those of both of us.

The infections were the usual ones found in the wards of a children's hospital. The routine surgical and medical methods were employed in addition to vaccine therapy. In attempting to determine the value of this form of treatment one encounters the same difficulties that are met with in deciding as to the usefulness of certain drugs, namely, that unless a marked specific result is obtained the estimate placed on their value is largely a personal one. And until a very large number of cases have been reported and statistics tabulated, with also a sufficient number of satisfactory control cases, a correct conclusion is very difficult. It is far easier to decide upon the effect of vaccines on local infections than on general systemic infections, for the reason that in the former the process takes place under one's eyes and any improvement is readily apparent. The incidence of local infections with pyogenic organisms is also immeasurably greater than systemic infections. General systemic infections go on unobserved with only the patient's condition to act as an index of the treatment. But what renders that judgment, at the present time, still more difficult is the improvement that has taken place in the technique of blood cultures. Modern technique now renders possible the recognition of a general infection which, a few years ago, would have been undiscovered. And this greater accuracy has been developed almost synchronously with vaccine therapy. It is now the habit to treat almost all such infections with vaccines, and therefore we have no proper control cases, that is, cases without vaccine treat-

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ment, to tell the outcome of certain forms of general infection accompanied by relatively few organisms in the blood. Several years ago, when the methods were such as to show only an intense invasion of the blood by microörganisms, the recognition of a blood invasion augured the most unfavorable outcome. The condition of affairs is different today, but what patients with a general septicemia can accomplish without bacterial vaccines is not sufficiently clear. It seems certain, from Libman's experience with streptococcus septicemia, otitic in origin, that if the infecting focus is removed the human body may rapidly overcome the invading organisms. The difficulty in deciding as to the location and the virulence of the infecting focus has much to do with increasing the difficulties of our conclusions.

We will discuss the cases which we have treated according to the organism producing the infection.

Staphylococcus Pyogenes Aureus, Local Infection:—

The infections by staphylococcus pyogenes aureus occurred most frequently as localized superficial abscesses. The abscesses thus treated were found on nearly every portion of the body from the scalp to the feet. In several cases there was general furunculosis. The larger number, however, were isolated abscesses occurring in various parts of the body. Of whatever etiology, the majority of these localized infections yielded readily to treatment by autogenous vaccines. The number of inoculations required was usually four or five, covering a period of two weeks.

The following case of general furunculosis illustrates the very satisfactory results accomplished by the use of autogenous vaccines:—

A rather undersized girl of eleven months developed a severe case of chicken-pox. The pustules were very numerous on the face as well as on the body. Following the disappearance of the pustules there occurred a general furunculosis. The abscesses were found on the thighs, buttocks and abdomen. They were deeply imbedded and apparently began in the subcutaneous tissue and gradually worked their way to the surface. A large number of these were opened, and from some as much as a dram of pus was evacuated. Surgical treatment was continued over a period of a week, during which time about fifty abscesses had developed and had been opened. A culture was then taken from one of the abscesses and staphylococcus pyogenes aureus isolated. A vaccine

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was administered every two or three days, five injections in all. Improvement was prompt and complete recovery rapidly ensued.

Staphylococcus Pyogenes Aureus, General Systemic Infection:—

Three cases are represented in our series and all 3 recovered. The clinical details of 1 of these cases is presented.

A child of five years was admitted to the hospital with a two weeks' history of tonsillitis, torticollis and swollen, painful joints. A few days before admission he began to have choreiform movements and gradually lost his power of speech. On admission he was not able to walk. Physical examination showed a faint systolic murmur at the cardiac apex; acute arthritis of the knee and elbow joints and constant movements of the face and extremities. During the first twenty days but slight improvement could be noticed. The temperature during this time was very irregular, varying from 104°F. to subnormal. Cultures from the throat and also from the blood showed staphylococcus pyogenes aureus. A vaccine was made and the first inoculation given on the twentieth day after admission. In all, five inoculations were given, covering a period of sixteen days. The child improved gradually under vaccine treatment; his temperature fell, all choreiform movements ceased and other manifestations of the infection disappeared. Without blood culture, it may be said that this case would have passed for a very severe chorea with unusual temperature, with no reason to suspect a blood infection.

Our experience with autogenous vaccines in localized staphylococcus infections has been entirely satisfactory. Their value can hardly be overestimated. In almost all instances they bring the process to an abrupt termination. Recurrences may take place, but they are again promptly controlled by inoculation. This has also been the experience of all who have written in regard to the vaccine treatment of such local infections. There can be no doubt as to their efficacy.

Very few general staphylococcus infections treated by vaccines have been reported. From our experience we are inclined to look upon such treatment with favor.

Streptococcus Infections, Localized:—

This organism has been isolated from the pus from localized abscesses, also from various cavities, such as the pleural cavity in empyema and from the middle ear in acute otitis media. It has been our experience that local infections due to the streptococcus

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yield much less readily to vaccine treatment than do similar infections due to the staphylococcus. But it is also our opinion that they may yield to a certain extent. After prolonged treatment, however, we have obtained no beneficial effects in some instances, as the following case illustrates:—

J. B., aged eight months, during the course of a mild nasal diphtheria without febrile reaction, developed enlarged cervical glands. Several of these went on to suppuration and were incised. Streptococci were isolated from the pus and an autogenous vaccine was made and administered. The use of the vaccine had no perceptible influence on the course of the infection. Fresh abscesses formed one after another in the cervical region, went on to suppuration and were incised. After a prolonged trial by autogenous vaccine a stock vaccine from a case of similar infection was substituted, but no beneficial results were seen after several weeks of this treatment. The child later succumbed to a pneumonia.

Streptococcus, General Systemic Infection:—

The following case illustrates one of streptococcus infection with a favorable termination:—

S. B., a boy ten years of age, was operated on for mastoiditis. Two days later appendicitis developed, for which he underwent operation. His temperature came gradually to normal. On the seventh day after operation a sharp rise to 104°F. occurred, and thereafter his temperature continued of a septic type. On the tenth day after operation a blood culture showed streptococci. A stock vaccine was used in small doses for the next three days with no apparent result. Fifteen days after operation an autogenous vaccine was administered and repeated daily for thirteen days. The temperature gradually came to normal and remained so. Coincident with the gradual fall of temperature there was an improvement in his general condition. Complete recovery ensued.

The following also is a case of general streptococcus infection with a favorable termination:—

R. C., a boy of eight, had tonsillitis. A few days later acute otitis media developed and the ear drum was incised. From the onset of these definite infections the child had a temperature curve of a septic type, with a rise often as hight as 104°F. On the fifteenth day after the onset a blood culture was made and

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streptococci were isolated. A week later a second blood culture was also positive for streptococci. An autogeneous vaccine was prepared, but on the day before the child received the first inoculation there developed in the right axillary region signs of a pneumonic process. On the following day, in spite of the pneumonia, the child appeared much better and the temperature was not so high as on previous days. The child was given his first inoculation on the day on which his temperature began to fall. The inoculations were repeated daily for five days. The temperature continued to fall and his general condition was greatly improved. After two weeks he was convalescent. Several inoculations were given at weekly intervals. A third blood culture, taken at the end of the treatment, was sterile. It is difficult in this case to say just what relation vaccine therapy had to recovery.

It is also difficult to tell, from the literature, what is the value of vaccines in streptococcus infections. There are not many local ones reported. It seems probable that almost all general infections with a favorable outcome are reported and few with an unfavorable termination. Further statistics are required for a definite conclusion.

General Pneumococcus Infections:—

In all, over 50 patients were treated. Most of them were small children with bronchopneumonia, but there were a number of older children with lobar-pneumonia.

Our experience has been confined largely to the use of stock pneumococcic vaccines, which were used in various ways; in small and large doses repeated daily, or at intervals of three or four days. We are not able to report any apparent beneficial results from their use, nor were there any unfavorable ones. In a few cases the pneumococcus was isolated and an autogenous vaccine made. In such cases we did not observe any better results than in cases without vaccine treatment.

Unusual Infections:—

We have treated a few cases of unusual infection with vaccines and with a favorable termination. One was a boy of nine years, who had sustained a compound fracture of the tibia and extensive laceration of the foot. The usual surgical measures were employed. The temperature was approximately normal for a week, when it rose to 104°F. The surgical conditions became worse and the child appeared septic. Amputation was then done

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at the knee. The temperature did not abate; pus appeared in the amputation wound and metastatic abscesses in the upper extremities. A blood culture was negative. The pus from the abscesses showed b. coli communis. An autogenous vaccine was made and administered twice a week for the following four weeks. The temperature gradually came to normal and recovery took place.

Another case was a boy of three months who had had erysipelas. Following the erysipelas many local abscesses developed. During the first week they involved the left knee joint, both hands and the right thigh. During the second week abscesses developed in the forearm and right leg. The pus from the abscesses showed b. mucosus capsulatus. An autogenous vaccine was prepared and injected many times. The abscesses gradually healed, and one which developed in the right elbow joint contained sterile pus. The abscesses finally healed entirely, leaving no permanent deformity except a subluxation of the left knee joint. Under observation the child gained 3 pounds in weight.

Both of these children seemed distinctly benefited, and their recovery due, in part at least, to the use of autogenous vaccines. It would be unwise, however, to attach too great importance to isolated instances of recovery following such unusual infections.

Gonococcus Cases:—

It is very difficult to obtain satisfactory data regarding the vaccine treatment of the vulvovaginitis of children; first, because such cases are not willingly received into hospitals and are always sent out at the first opportunity; and, secondly, because when at home they are liable to a reinfection from the source from which their original infection was received. There is also great difficulty in following such cases over the long period of time which must necessarily elapse before a cure can be reported.

The majority of reports in the literature are unsatisfactory because the observations were made in hospitals, and but few patients were observed subsequent to their discharge. There has also been no agreement among writers as to what constitutes a cure.

Butler and Long reported 25 patients treated, with no observations after discharge from the hospital. Churchill and Soper reported 41 patients treated, 9 of them observed at a later period; 3 of the 9 had relapsed, and of the 6, who had remained free from symptoms, only 4 were seen at a period later than two months.

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A. Hamilton and Cooke report 37 patients treated, but of these only 7 were subsequently observed. Four of these had relapsed within one week and the other 3 were not seen later than two months after leaving the hospital.

Thomas reported 13 patients treated, none of which were seen after leaving the hospital.

All of these writers believe that the discharge is lessened and that the gonococci are temporarily diminished or disappear. But an analysis of their figures shows that of 116 cases treated only 16 were subsequently followed (many for only a few weeks), and of these 16, 7 relapsed.

The latest and most optimistic addition to the literature on this subject is that written by B. W. Hamilton. He reports 84 cases treated by vaccines, with 76 cures and 8 failures. Nineteen of his cured patients were seen five months after the last evidence of infection had been present and were all found free from gonococci. These results are in marked contrast to those obtained by all other writers.

We have used stock gonococcus vaccines as a routine measure in the vaginitis ward of Bellevue, in conjunction with the usual irrigation treatment. Several cases in the out-patient department have been treated with vaccines alone. In many cases the combination of vaccine and irrigation treatment caused the discharge to be very greatly diminished or to disappear. But the period of observation was not sufficient to enable us to draw any conclusions as to the ultimate outcome. A few cases in the hospital treated until the discharge had ceased appeared in the out-patient department with a profuse discharge.

We have not been impressed by the efficacy of this form of treatment and believe, with the majority, that it improves symptoms but rarely effects a permanent cure.

DISCUSSION.

DR. CHURCHILL.—I would like to refer to a case of meningitis due to the staphylococcus, obtained three times from the spinal fluid by lumbar puncture, with large numbers of polynuclears. The case was twenty-one months old. The staphylococcus aureus was obtained on three successive punctures and an autogenous vaccine made up and injected, 70,000,000 dead cocci being injected subcutaneously. Temperature fell to normal and the child

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made an uninterrupted recovery. The history of the case is best shown by the temperature chart which I have here. It drops to normal immediately after the use of the vaccine.

In the case alluded to by the writer we came to the conclusion that the disease was shortened by the administration of the vaccine, but beyond that we did not feel that we could say anything about it. It seems to me that the staphylococcus vaccine is the one vaccine *par excellence* with which we do get results.

DR. KOPLIK.—My experience with vaccines has been limited to two infections—that of the streptococcus and staphylococcus. In the staphylococcus infections my results agree with those of Dr. Howland. With the autogenous vaccines, in cases of multiple abscesses, the results have been very favorable, and I always use that treatment now if I can get an autogenous vaccine. With the streptococcus our results have been rather discouraging, limited to a few cases in which the vaccine was injected subcutaneously, with little or no effect.

DR. SAUNDERS.—I would refer to a case of pyelitis, such as we see frequently in infants—a child of eight years, strong and robust; had had a number of relapses. An autogenous vaccine was prepared, pure colon bacillus, and injected. The condition was improved, but not cured. Finally we injected the vaccine, together with nuclein. Before that there was no reaction; with that there was pronounced reaction, vomiting, temperature 102°F., and erythema. Eight days later it was repeated and the temperature rose to 104°F., with vomiting and more severe local reaction, and then the case cleared up.

In Heath's last edition he refers to his method of using nuclein in conjunction with the vaccine to increase leukocytosis. Generally, when I have not had good results with the vaccine alone, I have employed the nuclein by mouth.

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THE BACTERIOLOGY OF THE BLOOD IN EARLY LIFE.

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Medical literature is full of reports of the bacteriologic examinations of the blood in adults, and much valuable information has been obtained therefrom. Various infections have been studied in this way; typhoid, pneumonia, endocarditis, erysipelas, osteomyelitis and many of the obscure febrile cases so frequently seen in our hospital wards. But while much work of this nature has been done among adults, we find comparatively few reports on this subject among infants and children. In scarlet fever alone have bacteriologic examinations of the blood been made to any extent. Study of this infection by Jochmann, Baginsky, Slavyk, Hektoen and others, has shown the presence of a streptococcus, apparently identical in its characteristics with the streptococcus erysipelatis. Thus, Jochmann, studying 161 cases of scarlet fever, found the streptococcus twenty-five times. All of these cases died, and Jochmann, from analysis of his own cases, and those of other authors, comes to the conclusion that the presence of this organism in the blood in scarlet fever gives an almost fatal prognosis. Curiously enough, however, neither Jochmann nor Slavyk found the streptococcus in the foudroyant cases also invariably fatal.

Aside from these studies on scarlet fever, reports on other infection in children are meager. Rotch reports a series of 36 cases comprising a variety of infection, the greatest number of any single infection being typhoid (8). He points out the scientific and practical value of taking blood cultures and urges a more widespread use of this method of investigation.

Otten reports a series of 250 cases of lobar pneumonia in children up to fifteen years of age, with blood cultures in 70. He found the pneumococcus in 9 of these 70 cases, 13 per cent.; of these 9, 4 recovered, 5 died.

The paucity of studies upon this important subject in children is undoubtedly due to the difficulty of obtaining sufficient blood

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for examination from this class of patients. The veins of children are so small that it is extremely difficult to get into them. We have ourselves experienced this difficulty, and besides the cases reported below have made numerous but unsuccessful attempts to obtain the blood from other patients, chiefly infants and young children. Others apparently have had the same experience. Indeed, one author, studying a large series of infections of otitic origin among adults, "throws up the sponge" at the outset and says that no attempt is made to study children on account of the difficulty of carrying out the technic. But, considering the frequency of obscure febrile infections among infants and children, it would seem that in such patients especially systematic bacteriologic blood examinations might be of great value; furthermore, in view of the future possibilities of antivaccine and antiserum treatment, accurate knowledge of disease is more than ever desirable; hence any method of investigation which promises to give us this accurate knowledge should be thoroughly tested and, if prolific of results, be systematically employed. It seems worth while, therefore, to report the following cases, even though small in number.

We have made 50 examinations on 44 infants and children during life, and 15 cases postmortem. It is with the former group of cases that we are concerned. This is comprised of the following infections:—

Typhoid	15 cases	10 positive results
Pneumonia	7 "	1 " result
Rheumatism	5 "	5 negative results
Meningitis (meningococ.)	5 "	2 positive "
Meningitis (staphylococ.)	1 case	1 negative result
Meningitis (tb.)	1 "	1 " "
Chorea	2 cases	2 " results
Pyelitis	1 case	1 " result
Erysipelas	1 "	1 " "
Bronchopneumonia	1 "	1 " "
Appendicitis, Gen. peritonitis, Lobar pneumonia	1 "	1 " "
Leukemia	1 "	1 positive "

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Thus, of the 50 examinations made on these 44 children, we have obtained 15 positive results. These will now be analyzed according to the individual infections.

Technic.—Most of the cases have been studied at the Children's Memorial Hospital, a few each at the Cook County and Presbyterian Hospitals, and to the attending physicians at these hospitals we are much indebted for the material used.

The blood has been obtained by the usual method, from the median basilic vein just above the elbow. In 2 cases of suspected typhoid it was obtained from the lobe of the ear, as recommended by Peabody. Various kinds of syringes have been used, but the Luer glass syringe has been the most satisfactory. The blood thus obtained has been mixed immediately at the bedside with various media. In most of the cases two tubes of bouillon and an agar plate have been inoculated. In 5 of the typhoid cases ox-bile has been used as the medium with a positive result in all. A case of suspected typhoid in which bile medium was used was negative, but proved later to be a case of tubercular meningitis.

In using the ox-bile as a medium, the following technic was observed:—

Test tubes containing fresh ox-gall sterilized in the auto-clave were used. The blood obtained from the vein, or from the lobe of the ear in some cases, was introduced directly into the test-tube of bile. This was then put in the incubator from fifteen to eighteen hours, at the end of which time a few cupfuls were transferred to a tube of coagulated blood serum. After three to five hours of incubation microscopic examination of the water of condensation showed, in the positive cases, the presence of motile bacilli. The presence of such motile bacilli is now accepted as evidence of a typhoid or a paratyphoid organism.

Typhoid.—It is in this infection perhaps that the greatest amount of blood culture work has been done among adults with interesting and important results. During the first week of the disease 95 to 100 per cent. of the cases will show the presence of typhoid bacilli in the circulating blood. With the progress of the disease this number diminishes, so that in the second or third week they are found much less frequently (40 per cent.). As the Widal reaction pursues exactly the reverse course, absent at first, present later, the blood and culture test becomes an extremely important method in the early diagnosis of typhoid, at least among adults.

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TABLE I.—ANTEMORTEM.

Case	Age	Sex	Clinical Diagnosis	Day of Disease	Number of Cultures	Medium	Result	Remarks
1	4 yr	M	Typhoid	?	1		Neg.	Widal incomplete 3 times, persistent leukopenia. Recovery
2	7 yr	M	"	10, 13, 20	3	Agar plate 2 Bouillon	All neg.	Recovery. Widal pos. on 10th day
3	12 yr	M	".	14	1	Bile	Pos.	Recovery. Widal positive
4	11 yr	F	"	6	1	"	"	Recovery. Widal negative on 4th day. Culture taken on 2d day of measles
5	3½ yr	M	"	7?	1	"	"	Ent. hosp. for genu valgum. Blood from ear on 3d day of a measles rash. Widal neg. 6th day.
6	5 yr	F	"	22	1	"	"	Recovery. Widal neg. 21st day
7	10 yr	F	"	?	1	"	"	
8	11 yr	F	"	10	1		"	Recovery. Severe case. Hemorrhages
9	6 yr	M	"	7th wk	1		Neg.	During relapse. Recovery. Widal pos. 8th day of disease
10	8 yr	F	"	14, 18	2		Both Positive	Recovery. Widal positive 15th day
11	5 yr	M	"	6	1		Pos.	Recovery. Widal positive 5th day
12	12 yr	M	"	8	1		Neg.	Recovery. " " 8th " Serum reaction to "para-colon" b. and to "para-typhoid" b. negative
13	11 yr	M	"	3d wk	1		Pos.	
14	4 yr	M	"	15 days	1		Neg.	Widal negative 15th and 17th day
15	3½ yr	F	"	8	1		"	Widal positive 8th day
16	1½ yr	M	Pneumonia	?	1		"	Second day of a high temperature which was probably a relapse. Outcome unknown. Child removed from hospital against advice
17	2½ yr	M	"	?	1		"	Recovery
18	7 yr	M	"	15	1		"	Lysis on 15th and 16th day. Recovery
19	9 yr	M	"	10?	1		"	Appendicitis; general peritonitis; pneumonia. Death
20	2 yr	F	"	8	1		Pos.	Rachitis Temperature normal 9th day. Convalescence tedious. Recovery in 2 months
21	3 yr	M	"	8	1		Neg.	Crisis on 9th day. Recovery
22	2 yr	M	"	4	1		"	Cretin. Death 4 hours after culture. No autopsy
23	16 mo	F	Broncho-pneumonia	2 wks?	1	Bouillon	"	
24	5 yr	M	Lobar pneumonia	5th day	1	Bouillon Agar plates	"	2 sisters and a brother, all in C. M. H. with pneumonia. Many pneumococci and few streptococci in cultures from deep sputum in all three cases No influenza bacilli. Typical signs in R. lung in all. Recovery in all.
25	7 yr	F	"	4th "	1	Agar plate	"	
	3 yr	F	"	11th "	0	—	—	

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TABLE I.—ANTEMORTEM (*Continued*).

Case	Age	Sex	Clinical Diagnosis	Day of Disease	Number of Cultures	Medium	Result	Remarks
26	11 yr	F	Chr. Endocarditis	?	1	Blood Agar, Bouillon	Neg.	Acute exacerbation. Culture taken 2 months before death on May 1
27	4 yr	M	"	3, 11	2	Agar Bouillon	"	During intercurrent tonsillitis (streptococcus)
28	6 yr	F	"	?	1		"	Culture on 2d day of a 3 days' temperature, without evident cause. Recovery from this
29	11 yr	M	"	months	1	Agar Bouillon	"	Antecedent history of rheumatism. Ent. C. M. H. on 3-19-10. Temp. 98°-103° from 3-27-10 to 4-7-10. Culture taken on 4-1-10. Temp. 99°-102° that day
30	11 yr	F	Chorea, Endocarditis, Chronic	7th wk	1	Agar Bouillon	"	No temperature. Recovery from chorea
31	10 yr	M	Chorea, Chr. Endocarditis	19	1	Agar Bouillon	"	Antecedent history of rheumatism. Culture taken after 3-4 days' temperature without evident cause, when T. normal
32	5½ yr	F	Rheumatic Fever	9, 16	2	Agar Bouillon	"	Chronic (?) endocarditis and arthritis, improvement; then measles, acute tonsillitis (strept. and staph.), general septicemia, death. Cultures during acute stage of the arthritis, etc. No autopsy
33	3 yr	M	Meningitis	5	1		"	Meningococcus in Sp. Fl. Death 1 month later
34	10 yr	M	"	?	1		Pos.	Meningococcus in Sp. Fl. Death 11th day
35	5½ yr	M	"	7	1		Neg.	Meningococcus in Sp. Fl. Death 51st day
36	9 yr	M	"	5	1		Pos.	Meningococcus in Sp. Fl. Recovery
37	6 yr	F	"	48	1	Bouillon, Agar plate	Neg.	Meningococcus in Sp. Fl. Death 4 weeks later
38	22 mo	F	"	12	1		"	Staphylococcus in Sp. Fl. Recovery
39	10 yr	F	"	10	1	Bile	"	Suspected typhoid. Widal negative. Von Pirquet positive on 10th day. Sp. Fl that of tb. meningitis. Death
40	20 mo	F	Pyelitis	weeks	1	Agar Bouillon	"	Rachitis, chronic endocarditis. Otitis media 2 days before culture, pneumonia 6 weeks later, death. Autopsy (postmortem case 13)
41	8 yr	F	Erysipelas	5	1	Agar Bouillon	"	Recovery
42	12 yr	F	Acute Nephritis	4	1	Agar Bouillon	"	Entered C. M. H. with typhoid symptoms. Widal neg. on 4th day; leukocytes 18,000. Culture on April 8 10; endocardial murmur on April 11, and pericardial friction-rub on April 12
				25	1	Agar	"	Acute arthritis, 5th day of temperature. Chorea
43	4 yr	M	*Acute Lymphocytic Leukemia	Several weeks	1	—	Pos.	Streptococcus found 1 week before death, also in heart's blood at autopsy
44	4½ yr	F	Pulmonary Abscess	17	1	Bouillon	"	Streptococcus found in pure culture

* Churchill.—*American Journal of Medical Sciences*, October, 1904.

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How is it among children? Our own cases are, of course, too few to warrant conclusions. We can state only that 10 of the 15 cases gave a positive result, and that these positive results were obtained as early as the sixth, and as late as the twenty-second day. (See Table I.) The negative results were obtained in the first case on the tenth, thirteenth and twentieth days, in the second case during the seventh week (relapse), in the third and fourth cases on the eighth day.

Combining Rotch's series, our own and a few scattering reports in literature, we find 26 cases of typhoid fever, in 19, or 72 per cent., of which bacilli were found in the circulating blood. We find, furthermore, that they were found as early as four days before the onset of symptoms and as late as the twenty-second day. The former case was a twelve-year-old child, whose sister was already ill with typhoid (Conradi).

These results were obtained with all kinds of media and at all periods of the disease. It is probable that with the more widespread use of Conradi's ox-bile medium and more frequent examinations early in the disease, this proportion of positive results will be considerably increased. Only when large numbers of cases have been examined, and at different times of the disease, can we draw final conclusions as to the importance of this particular laboratory investigation. If the results of the future correspond with the results already obtained in large numbers of adults, this method will become even more valuable among children on account of the more irregular, atypical nature of the disease in early life. We urge a constant, widespread use of the method, that our knowledge in this direction may be increased as rapidly as possible.

Pneumonia.—The pneumococcus is a frequent invader of the blood in adults, not only in pneumonia, but also in pneumococcic infections with the pathologic seat of the disease in other localities, *e.g.*, the endo- and pericardium, the joint, the meninges, the tonsils, etc. As in typhoid the literature is voluminous as to adults, meager as to children.

Otten studied 250 cases of pneumonia in children up to fifteen years of age. He made blood cultures on 70 of these patients, finding the pneumococcus in 9, or 13 per cent. Of these 9 children 4 recovered, 5 died.

Spitta describes a case of lobular pneumonia in a seventeen months' infant, with a general pnuemococcic infection, resulting

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in an arthritis and empyema; pneumococci were found in the blood during the sixth week of illness and one week before death. Autopsy confirmed the antemortem examinations.

We have taken cultures in 7 cases of lobar penumonia, and 1 of lobular pneumonia. The latter was negative. Only 1 of the 7 cases in the former group was positive. This was in a two-year-old female infant with rhachitis. The culture was taken on the eighth day of the disease; the temperature reached normal by lysis on the ninth day, the patient finally recovering after a tedious convalescence.

Thus in the 77 cases comprising our own and Otten's series, 10, or 13 per cent., showed pneumococci in the circulating blood, a striking difference from its frequency among similar infections in adults. This failure to discover this organism more frequently in the blood in children is due to one of two causes: either the technique employed in the cases already studied is faulty or the organism does not invade the blood frequently. The technique employed is apparently important, great discrepancies existing among the authors who have studied this question in adults. Thus Lenhartz, Schottmuller, Grimm and Joachim find pneumococci in the blood in only a fourth to two-thirds of their cases, whereas Procharska and Weins find them in practically *all* their cases. The former group of observers worked with solid media, blood-agar—the former with fluid media—blood-bouillon. To this difference in culture media, Otten attributes the varying results obtained by these workers, maintaining that the pneumococcus grows better on fluid media, and that consequently those who use such media will get a comparatively high percentage of positive results. He himself, in his work on children, used fluid media, yet got his results in only 13 per cent. of his cases. Consequently, if his reasoning be correct, the pneumococcus is not a frequent invader of the blood in the lobar pneumonia of children. However, here again the cases available for study are too few to warrant final conclusions, either as to the frequency of its appearance or as to its prognostic value when present. Future investigations must determine these points.

The Rheumatic Affections.—In this group we have included cases of endocarditis, arthritis, tonsillitis and chorea, these manifestations occurring either singly or in combination with each other. (See Table I.)

Postmortem examinations in cases of endocarditis have shown

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a varied bacteriology, streptococci, staphylococci, pneumococci, gonnococci, influenza b; pyocyaneus, all having been found in the heart's blood and in the valves in such patients. During life, in cases of septic endocarditis in adults, bacteria have often been found in the blood (Billings). We know practically nothing of this aspect of the subject in children, however. Our own group comprises 7 cases; all had chronic endocarditis; in 5 of the cases the cultures were taken during a febrile stage, due either to a tonsillitis, an arthritis or to an unknown cause, possibly an acute exacerbation of the chronic endocarditis. There were 2 cases of chorea, and in both of these the temperature was normal when the cultures were taken. Cultures were negative in all 7 cases.

Meningitis.—It was formerly thought that in epidemic meningitis the meningococcus rarely invaded the blood, but recent investigations with improved technic show its presence in the blood in about 25 per cent. of the cases. We have examined 4 cases, with 2 positive results. The tubercular case was examined on the tenth day of the disease as a suspected typhoid.

One case each of pyelitis, erysipelas and acute nephritis gave negative blood cultures. Catheterized specimens of urine from the cases of pyelitis and nephritis showed a streptococcus in the former and nothing in the latter. One case of leukemia showed streptococcus, ante and postmortem.

Postmortem.—Much more work has been done on the bacteriology of the blood of children postmortem than antemortem, and after death the blood often shows the presence of organisms in great variety. We have reports upon the bacteriology of the heart's blood in 15 cases, of which 11 showed positive results. These cases comprised the usual run of hospital patients —intestinal, pulmonary, bronchial, otitic, etc. (Table II.) Three cases of special interest were 1 each of the hemorrhagic disease of the newlyborn, of erysipelas, and of status lymphaticus. The hemorrhagic disease showed in the heart's blood the staphylococcus aureus. the b. coli and a few streptococci; the erysipelas showed the streptococcus in pure culture, and the status lymphaticus, the b. coli, the streptococcus and an unidentified diplococcus midway between the pneumococcus and the streptococcus.

Of the 4 cases with a sterile heart's blood, 3 were as follows: a pyelonephritis due to the b. pyocyaneus, an otitis with a general septicemia, but entirely negative postmortem results, and a meningococcic meningitis; the 4th case was an endocarditis, in

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TABLE II.—BACTERIOLOGY OF BLOOD—POSTMORTEM.

No.	Sex and Age	Clinical Diagnosis	Anatom. Diagnosis	Blood: Ante-mortem	Blood: Postmortem	Hrs. after Death	Remarks
1	12 d.	Hem. Dis. of Newly-born	Sept. thromb. of umbil. ves sels, numer-ous hem'gs. Meningitis	No Examinat'n	Staph. Aur. B. Coli. Few strept.	5	All organisms in cerebrospinal and peritoneal fluids. Staph. and b. coli only in pericardial fluid
2	F 8 mos.	Ileocolitis	Ileocolitis, Broncho-pneumonia	"	Streptococcus	3	Strept. in pericardial, pleural, peritoneal and splenic fluids. Staph. aur. in bile, splenic and pericardial fluids
3	M Infant	Rachitis	Enterocolitis, Rhachitic Osteochon dritis	"	*Smears:* Gram-poscoccus, and a Gram-neg. bacillus *Cultures:* Staph. aur. and colon b.	8	
4	M 4 mos.	Malnutrition	Ileocolitis, Broncho-Pneumonia, Adhesive Pleuritis	"	3 organisms re-sembling in all re-spects Strept. Pyog. Staph. aur. Diplo-coc. Lanceolat.	6	Same organisms from pus of lung
5	F 4 mos.	Gas·ro-enteritis	Gastro-enteritis, Otitis Media, Bronchitis, Meningitis	"	Negative	36	Cultures from cerebrospinal fluid showed the colon b.
6	M 3 mos.	Broncho-pneumonia	Broncho-pneumonia, Bronchiolitis, Cerebrospinal Meningitis, Patent Ductus Arteriosus	"	Staph. aur. Strep. pyog.	28	Cultures from cerebrospinal fluid showed: Diplococ. lan-ceolat., Staph. aur., Strept. pyog., B. fecalis alkaligones
7	M 6 mos.	Entero-colitis	Entero-colitis	"	Staph. alb.	30	
8	F 11 yrs.	Endocarditis (Case 26) Table I.		Negative	Negative	16	Bouillon only used postmortem
9	F 3 yrs.	Otitis, Gen'l Septicemia	Nothing ab-normal found	No Examinat'n	Negative	16	
10	M 14 mos.	Pyelo-nephritis	Pyelonephri-tis, Broncho-pneumonia, cloudy swell-ing of myo-cardium, liver and kidney	"	Negative	18	B. pyocyaneus, in pelvis of L. kidney, also b. coli. Staph. alb. from mesenteric gland. Staph. aureus from R. ureter. Staph. citreus and micrococ. tetrag. from pericardium
11	F 5 mos.	Entero-colitis	Entero-colitis	"	Staph. alb.	50	
12	F 18 mos.	Meningitis	Status Lymphat.	"	A diplococcus mid-way between the pneumococ. and the strept. pyog. B. colon streptococcus	1	No meningitis
13	F 20 mos.	Rachitis, Pyelitis, Endocarditis, Laryngitis, Lobar Pneu-monia	Rachitis, Ac. Nephritis, Vegetative Endocarditis, Laryngitis, Lobar Pneu-monia	Negative	Contamination?		Colon b. on trachea. Case 40. Table I.
14	M 3 mos.	Entero-colitis, Erysipelas		No Examinat'n	Streptococ.		
15	F 6 mos.	Meningitis (Meningococ.)	Purulent Meningitis	"	Negative		Meningococci in Sp. Fl., ante-mortem

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which the antemortem blood was also sterile. (Case 26, Table I.)

The organisms recovered postmortem were as follows: the staphylococcus, six times; aureus, five times; albus, one time; the streptococcus, six times; b. coli, three times; the diplococcus lanceolatus, one time, and the unidentified diplococcus already mentioned.

We are much indebted to Professor E. R. LeCount, of the Pathological Department, under whose supervision most of the postmortem work in our cases was done.

Summary.—The main points in this paper may be briefly summarized as follows:—

Blood cultures in adults in large numbers of cases have yielded information of scientific interest and diagnostic value.

Little work in this direction has been done in the infections of early life. There is need of more of this work among infants and children.

It is reasonable to suppose that blood cultures in early life may be of even more value than in adult life on account of the greater frequency of obscure infections at that time of life.

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DISCUSSION.

DR. HOLT.—The subject is interesting from many points of view. While of considerable value in older children, the taking of blood cultures in the infections of infants has not appealed to me strongly as a practical measure, to be freely used, especially in private practice.

In those cases in which I have had blood cultures made I must confess that they have given very little important information. There are other diagnostic measures which seem to me to offer more promising results and which are greatly neglected. The one to which I refer particularly is the taking of cultures from

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bronchial secretions in respiratory infections. The results of Dr. Churchill's work in pneumonia correspond very closely with our own findings, *i.e.*, that in pneumonia the organisms are not often found in the blood and that the finding of them does not help out much in diagnosis or treatment. In typhoid fever of course blood cultures have a distinct advantage for early diagnosis, but typhoid is not a common infection in infants. Blood cultures are especially valuable in protracted infections of obscure origin and in some of these nothing can take their place, but even in such cases of prolonged obscure temperature I would like to make a plea for a careful search for the bacillus of influenza, which may be the cause of many such temperatures. In a child recently seen who had had an attack of grippe, followed by otitis, such temperature continued; the otologist said a blood culture must be made on account of possible mastoiditis or sinus thrombosis, although the child at the time showed no prostration and was practically convalescent. I believe that the otologists have so many blood cultures made in adults that they are inclined to recommend them in young children when not necessary and they are likely to depend too much upon their findings.

DR. KOPLIK.—In regard to very young infants and children about two years of age, I have the same difficulty as Dr. Holt in getting enough blood to make a satisfactory culture, and in these cases the results have not been so gratifying as in older children. In order to get the blood we have to make an incision of considerable extent. Under these conditions we have made blood cultures in young infants and children, and I must say in regard to results that they have not materially aided us in changing our course of treatment. To instance one case—a newborn baby—we had to make quite a little incision; established streptococcus in the blood as the cause of the meningitis. Later on we established the typhoid bacillus in the blood of a very young infant who subsequently developed meningitis. In both cases the disease was fatal. And so I think we should hesitate before subjecting them to the test.

In older children we have made routine cultures where we did not get Widal reaction in typhoid, and found it very useful in the hospital, but I feel that in private practice we could scarcely recommend it in the first week of the disease. So that as far as the typhoid is concerned, it seems to me that at present it is only a matter of scientific value.

We have taken cultures in cardiac disease, with recurrent temperature; our idea being to try to establish the fact that there was an infective endocarditis. Out of quite a large number of cases we have only discovered 1 case of chronic-infective endocarditis, and that was where I least expected to find it; a little girl, eight years old, running normal temperature when she came in, then her temperature went up to 100.5° F. We found a streptococcus

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of very greatly attenuated virulence in the blood. The interesting part of this was that the child really developed, after the culture had been taken, petechiæ, and all the signs of infective endocarditis. It was very difficult to make a vaccine from this attenuated streptococcus, as we could not cultivate it. I mention these facts to show that there are many sides to this question of making blood cultures in infants. I had one case where the otologist had operated on the ear after an attack of grippe, then from the ear to the mastoid—double mastoid—and they were considering seriously opening both sinuses. Now a child can scarcely stand such operations. I would warn about conclusions drawn from findings in these cases; sometimes there is an obscure trouble which cannot be located. On the other hand, there are cases with brilliant results. A boy nine years of age came in with a simple history of having had an otitis a week before. The drum was opened; closed and there was nothing to be seen in the ear; the boy ran a pretty high temperature; for a few days 105° F. to 106° F. up and down. A culture of the blood was taken and streptococcus found. The boy looked well and belied his temperature. They decided to cut down upon the sinus and doing so found an old thrombus, white, and partly broken down. There was absolutely nothing in the mastoid nor in the ear.

DR. ABT.—It seems to me it makes a good deal of difference who takes the blood culture. There are blood cultures and blood cultures. I have seen men bungle around and make a large wound. There is some advantage in encouraging men to study the technique of this work in order to perfect it. I think in our hospitals we should make these cultures and I have found that they throw a very important light upon many conditions. I recall a case of obscure disease in which the child went down and down, with increasing anemia, and when a blood culture was taken the blood was found to be swarming with streptococci.

It would be valuable if men making blood cultures would along side of that make urinary cultures also.

DR. BOVAIRD.—I would like to say just a word in favor of the use of blood cultures even in children. We should remember that our methods are not complete or final as yet. It may be true after a while that where we require now 10 c.c. to get a satisfactory culture, the pathologist will be able to give us a satisfactory reply with a much smaller supply of blood; in fact, even now some pathologists do this; even from a very small amount they get satisfactory results. Perhaps the method is not yet perfected that will enable them to do this with a few drops of blood, but it may be before long. A dissection is surely not necessary in all cases for obtaining blood. One who has had experience with intravenous injections knows it is quite possible to obtain the blood in many cases by puncture of the vein with a hypodermic needle. If the pathologist can perfect his method so that only a few drops

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are required, we may be able to get the necessary amount with a hypodermic needle.

As to the results of the method, it is true of course that thus far they have not yielded much in children; but we know that they yield results of vast importance in adults. In the matter of typhoid fever alone, we can now make a diagnosis with a certainty that was impossible a few years ago, and for the differentiation from typhoid of the irregular fevers that are seen in hospital wards so frequently it is essential. If we are to approximate in any degree the same sort of accuracy in the treatment of diseases of children we must follow the same methods and every contribution along this line is of very definite value in adding to our exact scientific information and in helping us to an understanding of the diseases of childhood.

DR. EDSALL.—I have had very little experience with infants for some years past, and hence have no information of importance in regard to the use of blood cultures with them. In older children, however, I have for years employed blood cultures not infrequently in obscure cases and have come to rely very greatly upon them as one of the most valuable means of diagnosis that we have in certain difficult conditions in which there is a possible infection. I must disagree entirely with Dr. Koplik's statement that blood cultures are purely of scientific value in typhoid fever. I have in many instances been in great doubt as to the nature of the condition that I was dealing with, when blood cultures have helped me more than anything else. I quite agree with Dr. Holt that blood cultures should not be used in cases in which a diagnosis can be made in easier ways, but nevertheless I think that cases are not uncommon in which they constitute the most important method that we have of showing the actual presence of a general infection and the nature of that infection, and certainly a definite knowledge of what we are dealing with is the first step toward accomplishing something for the patient. I feel very seriously that it would be unfortunate to speak of blood cultures as constituting a method of little value.

DR. CHURCHILL.—Dr. Bovaird and the Chairman have expressed my views pretty well on the subject. I would not give the impression that I think it of value in infants alone; in infants it is apt to be of less value than in older children, but there is no reason why we should abstain from trying to perfect the method. It is likely that in the future the technique will be so improved that it will be very much easier to do it. In regard to the technique I certainly do not advocate any dissection, and in only one case was the blood obtained in that way, and that by an enthusiastic interne. Unless there is likelihood of some very definite information being gained, of course we are not justified. The work should be done by an expert; Dr. Clarke did the actual work in these cases.

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We do not know enough about the subject of the bacteriology of the blood at present to draw any definite conclusions, but we must go ahead and study it. In view of the possibilities of vaccine and serum treatment the knowledge gained may be of great value. I have had an illustration of making a diagnosis in these obscure cases recently in a five-year-old child who came in with an obscure history and very obscure symptoms which all the methods of laboratory diagnosis failed to explain. The sputum was examined, the urine was examined and the blood was examined; everything we knew of was done. The only definite thing we could arrive at was that it was a case of general septicemia, and we found the streptococci in the blood. It did not in that particular instance do any good and it later on developed that the child was getting up a pulmonary abscess. Had we found the staphylococcus we would almost certainly have obtained favorable results with a staphylococcus vaccine. It seems to me we know little enough anyway about diseases in children and any accurate method that may add to our knowledge should be given a thorough trial.

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THE VALUE AND LIMITATIONS OF THE EMPLOYMENT OF COLD AIR IN THE TREATMENT OF ACUTE RESPIRATORY CONDITIONS.

By J. P. Crozer Griffith, M.D., Philadelphia,
L. E. LaFétra, M.D., New York,
and Others.

OPENING OF DISCUSSION BY DR. J. P. CROZER GRIFFITH.

Dr. Griffith.—In entering into the discussion of this subject it is important to formulate a clear idea of just what is meant by the "cold air treatment," and the question raised whether we do not mean rather the *fresh* air treatment. Cold air may be conceived of as as acting in several different ways. First, it may reduce the temperature of the body suffering from fever. In this manner its action will be much like that of the cold bath. But, inasmuch as the person treated in the cold, open air is very carefully wrapped up and protected from chilling, this action is evidently not the one desired or obtained. Secondly, there is the possible effect of the breathing of cold air. Whatever the reflex action may be of the cold air upon the nasal and laryngeal mucous membrane—and I do not think that this is of great importance—it is evident that the cold air can have no direct effect upon the lungs, inasmuch as in the ordinary process of breathing the air is warmed before it reaches these organs. Thirdly, there is to be considered the breathing of *fresh* air, *i.e.,* constant out-of-door exposure to the air, whether this be warm or cool. This last is the subject to which so much attention has been given. The fact that the cool air of winter employed in this way has more benefit than the very hot air of summer is not, I think, due to any difference in their character as inhaled, but dependent upon other conditions, such as the general depressing effect of heat upon the system.

The value of this fresh air treatment is undoubted, but it is to be borne in mind that the treatment is in no way new. Its employment for phthisis particularly has within the last few years undergone a very wide extension, but it has been used for this purpose for many years. More recently it has been applied also to other respiratory diseases, even in children, as especially in pneumonia; and it is of undoubted value also in many other conditions, notably malnutrition in infants, and the like. Did time permit I could refer to many cases in my own experience which would duplicate, undoubtedly, others in the experience of all of you—several children, for instance, in different families, whom I have over and over again treated for colds in the heads and

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throat for years, until they commenced to sleep out of doors, when their tendency to colds disappeared. One of the most interesting instances, however, was the following: A little child of about two and a half years of age, with empyema, was operated on in the hospital of the University of Pennsylvania. He failed to gain in health until he was given the open-air treatment in the sun-parlor, when his progress was rapid. For some reason it was necessary to place him again in the ward, when retrogression of health commenced until I was able again to have him return to the sun-parlor, when improvement at once began again.

Useful, however, as this treatment is, I am convinced that it can be overdone, and there is, I think, danger that in our natural enthusiasm for what seem to be new and useful procedures we may lend to this a too unqualified approval. Our cases should be carefully selected. Unquestionably many delicate infants do not well bear exposure to very cold air. Some children with bronchitis, for instance, are undoubtedly helped more by confinement to rooms with very uniform and rather warm temperature, in the manner which was considered the only way some years ago. I do not mean, of course, that the air in these rooms shall not be changed with sufficient frequency. Often, too, bronchitis will be more rapidly cured by such procedure combined with the filling of the room with water-vapor, medicated or otherwise. This is an experience which has repeatedly come to me.

As I have intimated, it is often difficult to tell just which case should receive one plan of treatment and which the other, and, indeed, the choice can be often only empirically made. On the whole, however, I believe that the exposure of children with respiratory diseases to open air, whether warm or cool, is a very great addition to our treatment of disease in early life.

DR. L. E. LAFÉTRA.—In the discussion of this topic it would be of the greatest assistance to know the physiologic action of cold air upon the various parts of the respiratory tract, not only directly, but also reflexly, through the medium of the nerve supply of the face and the nares. Unfortunately, I have been unable to find anything written upon this phase of the subject. The direct effect of the cold air inspired through the nostrils can hardly go further than the back of the pharynx and upper aperture of the larynx, being warmed to body temperature and saturated with water vapor by its passage through the nasopharynx. The primary effect of the direct application of cold to the skin is to stimulate the contraction of the blood vessels and lessen congestion of the parts; prolonged application has the secondary effect of producing relaxation and stasis. In the breathing of cold air, as compared with warm, there would seem to be only the difference of the greater amount of heat and moisture required in the nostrils for raising the inspired air to body temperature and saturation. There is no prolonged application of cold—except, perhaps, to the face—since with each expiration there is the

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passage of warm, moist air over the nasal mucous membrane. With the remainder of the body kept warm, the cold air can affect only nerves of the face and nasal mucous membrane, and in this way reflexly the circulation and the caliber of the bronchial tubes and also the muscular tone of the larynx. The dryness of the air also has an effect as far as the larynx. Clinically, we know that the inhalation of warm, moist air relaxes the larynx and the bronchial tubes and promotes secretion; we know clinically, also, that cold, dry air, in the acute congestive stages of laryngitis and bronchitis is apt to produce spasm of the larynx and contraction of the bronchial tubes.

For the purpose of gaining all the advantages of the tonic effect of cold air in the treatment of hospital patients, Dr. Holt established, three years ago, at the Babies' Hospital, a "cold ward." The temperature in this ward was kept at about, and not over, 50°F., from November to April. Next to this cold ward is a regular ward, kept at a temperature of 68° to 70° F. In connection with this ward there is a steam room for administering vapor inhalations. Both wards have ten beds and have southern exposure. Into the cold ward have been put especially cases of lobar pneumonia, bronchopneumonia, empyema, pulmonary tuberculosis, bronchitis; also cases of rickets, malnutrition and of anemia. The number of cases in the cold ward was 50 the first year, 62 the second, 67 the third and 20 the fourth, this last number being smaller on account of the development of measles in the ward. Altogether, about 200 cases of the general classes mentioned were treated in this ward. There would be no advantage in detailing the statistics of the cases, nor of their mortality, since the cases differed so greatly. More important are the impressions gained by watching the effect in individual cases.

The advantages of the cold ward as regards the pneumonia cases are most marked in reducing the irritability and restlessness, promoting sleep, lessening the dyspnea, with the lowering of the respiratory and heart rate. These effects were most marked in cases of frank lobar pneumonia with high temperature and little or no bronchitis; less marked in bronchopneumonia. The good effects on the feeding and anemia cases were seen in increased appetite and improved assimilation.

Cases that had developed laryngitis or sibilant piping râles were not benefited, and with 3 patients in particular removal to the warm ward with or without steaming was followed by distinct improvement. These cases were the following:—

CASE I. Annie B., thirty-two months, bronchopneumonia, admitted December 28, 1909, to the cold ward, temperature, 50°F. The dyspnea increased so that same evening the child was transferred to the adjoining warm ward, temperature 70°F. The physical signs in this case were some dullness of right lower chest, with feeble breathing and very abundant fine, moist râles. There was considerable dyspnea. Child was cured in five days.

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CASE II. James K., nine months, admitted December 24, 1909, with bronchopneumonia, temperature 103° to 105° F.; sent to cold ward. Kept in this ward for five days, but breathed with difficulty, so was moved to warm ward and steamed. The change was followed by improvement, but the child developed a retropharyngeal abscess, otitis media and tetany with laryngismus, so he died two weeks later.

CASE III. V. B., postoperative empyema. The child was put in cold ward, but got very blue. Removal to warm ward was followed by improvement. Here, perhaps, the cold air sucked into the chest through the wound may have had an influence.

Another case with influenza bronchopneumonia was moved back and forth twice from cold ward to warm ward with no apparent change.

From the cases that have been treated in the cold ward and in the adjacent warm ward the following conclusions may, I think, be drawn:—

(1) In the acute congestive stages of inflammations of the upper respiratory tract (rhinitis, laryngitis, bronchitis, especially of the small tubes, with or without bronchopneumonia) warm, moist air is of greater help than cold, dry air.

(2) In lobar-pneumonia, with high temperature and little or no bronchitis, cold, dry air is of great advantage. The same is true of pulmonary tuberculosis and of empyema.

(3) After the acute stage has passed, and when there is no inflammatory spasm of the larynx or bronchi, the cold-air treatment is of advantage in cases of rhinitis, laryngitis, bronchitis and bronchopneumonia.

DR. NORTHRUP.—When you talk about cold air it must be remembered that cold air is not necessarily fresh air. The air in the cellar is cold but not fresh. On the roof of a hospital the air is fresh. We built a shelter because of the storms from the west, and the first thing the nurses complained of was that the air was not good; they said they could not breathe well. It is because it is still air. The essential of fresh air is partly that it shall be circulating, moving air.

DR. GRAHAM.—Looking at the matter from the standpoint of private practice, I think one is quickly convinced that unless you get cold air in the sick room you do not get fresh air. In other words, in a private house if the room is warm fresh air is not being admitted. In hospitals we can regulate conditions exactly and have just as many windows open as necessary, but in private practice the room that is not cold is apt to contain vitiated air. It is necessary in private houses to have the room cool, at least 60°F. or below.

DR. HOWLAND.—It is, I think, the almost invariable experience of every one that children with acute infections do better in cold air than in warm air, and it is also, I think, true that chil-

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dren with infections, especially pneumonia, do better out of doors in winter than in summer. It was shown years ago by the researches of Romberg and Pesler that the cause of death in acute infections was to be ascribed first to paralysis and later to collapse of the vasomotor system, and not of the heart. It was shown years ago also that with the use of vasomotor stimulants the blood pressure could be raised with amelioration of symptoms and cure in cases that otherwise would result fatally. There had been no attempt to do this with children until last year, when Hoobler and I tried using various vasomotor stimulants. We found that children with pneumonia put out doors and then brought in and given caffein and camphor, which ordinarily causes a rise, would cause a fall. We tried the same thing with children out doors and then brought in, without stimulant, and found in almost every case that the blood pressure promptly fell when the child was brought into a warm atmosphere. This, I believe, has been the case without exception. The experiment could be varied at will. It is true of healthy individuals in the majority of cases. It is true in all cases of pneumonia, and the results are more marked the sicker the individual. It is known that in the acute infections, as dysentery, typhoid and tuberculosis, there is a loss of blood pressure from the start, and the sicker the individual the greater the fall. Consequently, it seems to me a rise in blood pressure, which can be accomplished so easily as by putting the patient outdoors, can only result in benefit. The rise in blood pressure is permanent and constant and accomplished without disturbance to the individual at all. (The speaker showed results graphically portrayed by charts of some of these experiments; one child with blood pressure 95 mm. outdoors, brought in and blood pressure began to fall at once and continued until, at end of seventy-five minutes, it had fallen 15 mm., and continued low as long as the child remained indoors. A second child showed the same thing. A third child showed blood pressure of 130 outdoors and indoors a fall to 85. A fourth child showed blood pressure of 90 outdoors, with a fall of 10 mm. when brought in. The child was put out for six hours and had blood pressure of 102.5. A fifth child showed outdoors pressure of 100, brought in and there was a fall of 15.)

It is very interesting to see how this lowered blood pressure may be controlled. If you keep the child indoors and give caffein hypodermatically the blood pressure rises, but it will rise very little above a point which it would reach if the child were outdoors without the caffein. If you keep the child outdoors and give caffein the pressure will rise but little above that obtained by keeping the child indoors and giving caffein. The greatest and steadiest rise is accomplished just by the employment of cold air alone. It is very well known that stimulation of the skin and of the mucous membrane of the nose will cause prompt rise of blood pressure.

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DR. BLACKADER.—I agree with the last speaker as to the value of increasing blood pressure in the latter stages of the disease; in the early stages it would seem that lowering the blood pressure would be indicated. The reason I would use caffein or camphor would be to stimulate the respiratory center. I am an advocate of fresh, free, flowing air, but it is because I feel that the child is getting more oxygen and a certain amount of stimulation of the respiratory center, rather than because of increased blood pressure. A few years ago it was the practice to lower blood pressure by the use of nitroglycerin in the acute stages of the disease, lowering the blood pressure by bleeding into the large abdominal vessels. I cannot think that it is quite the point to be emphasized, although extremely valuable and interesting to know that we can influence blood pressure in this way.

DR. SOUTHWORTH.—I should like to emphasize a little the advantage of cold, that is, the lowered temperature, not as bearing upon the treatment of acute respiratory diseases so much as in their prevention. It so happens that during the past two winters we have been carrying out the fresh air plan in the wards, and we have done it by discontinuing the use of one of the large steam boilers, so that we now have but one, and it is impossible to get the temperature up to the heights it used to reach. The temperature of the wards now is often so low that the faces and hands of the children look rather blue and chapped, yet acute respiratory infections have become so rare among the inmates of the hospital that we practically only have the infectious colds, which can now be distinguished from the other types, as when they appear in any of the wards they go from patient to patient.

DR. HOWLAND.—I believe it has been well shown by Romberg and Pesler that the cause of death in acute infections that they studied, pneumonia, diphtheria, etc., and by the work of Ravinski also, is paralysis of the vasomotor centers and that the heart is practically competent throughout. With proper resistance offered to the heart's action, with use of barium chloride, or constriction of the aorta, the heart would go ahead and beat for hours. The best explanation of the gradual fall of blood pressure is the gradual effect upon the vasomotor centers. It is, to a certain extent, a hypothesis perhaps. Often respiration ceases before the heart, not from disease *per se* of the respiratory center, but because the blood pressure is so low that the vasomotor center is not supplied with enough blood. The heart goes ahead and beats long after cessation of respiration, trying to fill up the empty arterioles. I think it is generally believed now that the old observation that the heart was beating too hard at the beginning of the disease, and that administration of aconite was indicated, was due to poor observation. That the vessels are empty is due to the effect of the poison on the vasomotor center, and the heart has to overwork to compensate for this.

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DR. ABT.—Would you think of treating a patient with shock and collapse with cold? What kind of blood pressure apparatus did you use?

DR. HOWLAND.—I should certainly hesitate to apply cold to a patient in a condition of shock. The most striking result I ever saw obtained therapeutically in pneumonia was in a child that had lain for an hour or more pulseless absolutely. She was the color of death and the respiratory center had been so affected that she would breathe three or four times and then stop for four or five seconds. Her heart was beating violently. I did not hesitate to apply ice cloths to the abdomen and gave caffein and adrenalin hypodermically in enormous doses and she gradually came back so that her pulse could be felt and the respiration became regular, and in two hours she was conscious and she went on to recovery. Her temperature was 106.5°F. The patient was eight years of age. In surgical shock, I do not know.

We used the Fort apparatus, with a special cuff made much narrower than usual.

DR. GRIFFITH.—I would emphasize again that the sun parlor of the university has its windows open, and that the children are usually there instead of in the wards. That expresses my practice. What I have heard this afternoon, however, confirms me in my opinion that we are yet too far at sea regarding our knowledge of the actual relationship of physiology and of experimental therapeutics to symptomatology to permit us to confine ourselves to any one line of treatment. We are making a mistake if we abandon things we did ten years ago merely because they are old; we should be convinced that they are wrong. If there were time I should like to remind this Society of the various earnest discussions we have had in the past over the advantages, or the reverse, of various practices in which we were deeply interested at the time—the sterilization of milk, the pasteurization of milk, the value of cereal decoctions, the relationship of food to scurvy, and the like. There has always been a certain swinging of the pendulum and a final settling down to a certain quiet, defined position. So, too, as regards the value of cold air in the treatment of respiratory diseases. I am a firm believer in the method. Yet, are you satisfied that the employment of vapor in the room, the careful guarding against exposure, and similar practices in which we all believed a few years ago, had no value in any instance and did no good? I am not, although I admit I have drifted away from these methods on the wave of popular enthusiasm for the newer treatment. There is, I think, a little danger that we may make cold air treatment a fad, valuable as it undoubtedly is.

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THE NEED OF MORE COMPLETE REGISTRATION OF BIRTHS.

BY J. H. M. KNOX, JR., M.D.,

Associate in Pediatrics, The Johns Hopkins University, and President of the American Association for Study and Prevention of Infant Mortality, Baltimore.

I make no apology for bringing the subject of birth registration before this distinguished body, for it is one in which the Pediatric Society should be deeply interested. Our membership is made up of those who in this country are supposed to have most accurate knowledge of the physical necessities of the infant and of the factors influential in its individual weal and woe, and it would seem that, possessed of this knowledge, the American Pediatric Society has a peculiar responsibility to take a leading part in the rapidly spreading movement to lessen the influences which are at present operating before, during, and throughout their first year to diminish the vigor and destroy the lives of so many infants. We know as do few that a death-rate of 20 per cent. under one year can be reduced at least one-half by properly directed effort, and this very knowledge carries with it an overwhelming duty to the communities in which we live and to the country at large.

The investigation of infantile disease in the laboratory and at the bedside should lead to better methods of preventing illness and death among all babies. It is quite possible, however, that one who is fully occupied in private work may fail to see in his personal experience the needs of the infants in the whole city. Some knowledge of general statistics is necessary, therefore, to revise the impressions that we receive from a limited horizon.

Accurate information concerning an epidemic of measles, for example, is of more importance to the public than the results of any one man's experience with the disease. Knowledge of the incidence of and mortality from various ailments can only be derived from a study of general vital statistics and cannot be acquired by collating the recollections of a single life or institution.

This being true, we must face the fact that we have no complete national vital statistics. For only about 55 per cent.[1] of the population in eighteen states is there any registration of the

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number and cause of deaths. In no single state or city in this country is the registration of the births complete, and in but a few isolated states and cities is there any law on the subject which is properly enforced. The United States Census Bureau calls attention to the fact that elaborate and carefully worked-out systems record the birth, entire career, and death of every pedigreed horse, cow, and even blooded cats; yet children are born, and men and women die, without the slightest official record of these events being preserved. This indifference to record-keeping is perhaps to be expected in a new country in its formative period, but surely the United States has long since emerged from this chaotic condition. Its influence is said to be powerful among the nations of the world, and yet it is neglecting a very fundamental quality of self-respect, a knowledge and appreciation of its own most valuable possession—the lives of its citizens.

Vital statistics which reach a high standard of excellence are published each year from the following countries:[2] England and Wales, Scotland, Ireland, Australia, Tasmania, New Zealand, Ceylon, Jamaica, Denmark, Norway, Sweden, Russia, Finland, Germany, Austria, Hungary, Roumania, Bulgaria, Servia, Netherlands, Belgium, Switzerland, Spain, Italy, Japan, Chili; and to this list should probably be added Mexico, the Argentine Republic, and other countries in South America. The United States as a whole is not represented in international vital statistics. The difficulties are partly inherent to our form of government, and their solution will depend upon a thorough understanding between the several states or some uniform federal regulation. Behind all this, however, lies the persuasive power of public opinion, long dormant and unresponsive to this important subject. Until the public learns that it is essential for the best well-being of any complex civilized community to know the number, character and condition of its citizens, the fewer laws the better.

Some states not belonging to the registration area have had half a dozen general laws for collecting vital statistics, any one of which could be known to be a certain failure from the time it was enacted. As Professor Reinsch remarks,[3] "the amount of legislation in the several states as compared with that in other civilized communities is astounding. Many laws are passed which do not have a strong public sentiment behind them, or the enforcement of which is not properly provided for."

This paper is not a brief for *more* law, but a plea that we may

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aid in convincing the public that to sustain our self-respect among the nations as well as for other reasons, some of which may be mentioned, there must be an official permanent record of every birth which occurs in the country. From the legal point of view adequate registration of births is often of great value. It is frequently necessary to establish the date of birth of an individual before he can obtain certain property or secure a pension. It may determine the direction of an inheritance. In some states it may be needed to establish the right to vote, for jury, army, or navy service, for marriage abroad, often for even residence abroad, and to prove the age of consent (I am told that it is very difficult to convict a man accused of offence against a girl whose age cannot be proved by a birth certificate), to determine the real age in respect to child labor laws or school attendance, or in some cases to establish an identity. These are but a few of the many reasons why the lawyer would welcome uniform and complete birth registration. In lieu of this definite information all sorts of substitutes have to be made use of. One pension claim was established because there was a record of the foaling of a noted race-horse synchronously with the birth of the claimant. Many other probably just claims have remained unpaid because there was no positive knowledge of the time of birth.

"There is hardly a relation of life,"[4] says Dr. A. R. Reynold's, "from the cradle to the grave, in which the evidence furnished by an accurate registration of births may not prove to be of the greatest value. As the country becomes more densely settled and the struggle for existence sharper, many of those matters which hitherto have been of minor significance will take on deeper meaning and acquire greater importance. Hence the urgent necessity for remedy of the defects which prevent a proper registration of births."

Dr. Fulton[5] points out that "the interest of the citizen in registration of births is indeed superior to his interest in the registration of deaths, for a greater proportion of the privileges and immunities, rights and duties, turning upon the question of his age and his parentage, are definitely conserved by the registration of his birth."

But apart from and in addition to the importance of this matter from the legal point of view, there are even more urgent reasons that prompt registration of births with the proper authorities should be championed by every physician, particularly by those

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most interested in child life. I may mention several. First, accurate birth registration is the only means we have of determining the number of babies under a year in any community, and until we know this our discussion of the tremendous problem of infant mortality is more or less successful guesswork. Nearly all the writers in this country who have discussed the question have based their deductions upon foreign statistics or upon those of a comparatively small number of cities or communities. The latter, in the opinion of the experts at the Bureau of Vital Statistics, are very unreliable. The same is true of the census returns. The infant birth rate in the United States is not known, and hence the number of the infant population can be the subject only of conjecture. Such questions as these await solution: What proportion of infants born die within the first week of life? How many die of over-lying, of neglect, of gastrointestinal disease, from any cause, in proportion to their whole number? We do not know. In every other civilized country this vital information is at hand for the asking. Here we must imaginatively generalize from a paucity of statistical facts, or draw upon our own limited experience.

Again, would it not be well to have a prompt record of births, that the authorities could detect and treat ophthalmia neonatorum ere it is too late, and so prevent many of the cases of blindness that are crowding the asylums? More than half of the babies in this country are ushered into the world by midwives, but few of whom have had any training in surgical cleanliness. Their cases particularly need official oversight.

Again, it would not be nearly so difficult to get in touch with the mother, to encourage breast nursing, advise as to proper clothing, and adequate ventilation for the baby, if notification* to the proper authorities followed immediately after its birth. Many of the infants at the various medical and milk dispensaries come for the first time after weeks of illness, much of which could be avoided if the news of the arrival had been known.

If we have any duty to the children born out of wedlock, which so greatly swell the institutional mortality rate, it can be best performed by insisting on speedy notification, followed by competent advice and assistance. France first discovered the diminu-

* Notification of the fact of a birth at a definite address is to be distinguished from the fuller registration of the birth for statistical purposes, giving the child's name, that of the parents, their nationality, etc. The former should be done on a postal within a few hours, the latter within a few days. Both are necessary—one helps the other.

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tion of her population through birth registration. Again, is it not of the utmost importance in this country, whose population is made up of so many mingled elements, to be conversant with the vital statistics of each race in its new environment, to know the birth rate and the death rate, and the cause of death for every type of population, whether Irish, German, Slav, or African, Jew or gentile, native born, and in all parts of the country, inland or coast, prairie or mountain, and under differing home and industrial conditions and walks in life? With all these facts the science of demography has to do, and it depends upon birth registration as its very foundation stone.

At the rate of progress we are making, according to Dr. Cressy L. Wilbur, our best authority on this subject, we can hardly expect to secure complete birth registration covering the whole country until the year 2,000, but surely we shall not be satisfied with such delay in obtaining such facts, so essential to our national self-respect and to the well-being of our citizens. Already a forward step has been taken by the Bureau of the Census operating wth a committee of the Public Health Association in the drafting of bills[4] containing the essential requirements of registration for submission to the several states. It is hoped that this will prevent the waste of effort in enacting useless laws, as well as to supply a guide for efficient legislation.

Some of the necessary provisions for registration of births are:—

(1) They must be registered immediately after their occurrence.

(2) Certificates of birth should be required.

(3) Some check is necessary to secure enforcement of the law. A fine for failure to comply, and a reward for the officers detecting the delinquent.

(4) A central registration office and efficient local registrars.

(5) The responsibility for reporting births should be fixed.

It is the fact of registration and not the nature of the machinery by which it is obtained that is the goal to be reached. Certainly physicians have been most recreant in their duty. In Baltimore, in spite of a fairly good law, not more than about half of the births are reported. If the relationship between the number of deaths and the number of births was as statistically reported, our city would be gradually depopulated. Possibly in many communities better results could be obtained if the Liver-

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pool plan were adopted and the responsibility laid upon the father of the child if residing in the house, or upon the person attending the mother. This person is simply required to notify the authorities of the *fact* of the birth within six hours through blanks and postage furnished. The remaining data is obtained by the health officer. Surely the subject is one of the first importance for many considerations, some of which have been touched upon. No body of men exert more influence to secure adequate birth registration in this country than the members of this society. When we follow the obstetrician in the treatment of the newly-born infant, let us see that its birth is duly recorded, and in general, as experts and as public-spirited citizens, let us labor to remove this reproach to the intelligence of a great nation.

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ON ACUTE PERICARDITIS IN CHILDREN.

BY DAVID BOVAIRD, JR., M.D.,
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Acute inflammation of the pericardium, while not a frequent lesion in childhood, is not uncommon. The subjoined charts illustrate this statement in effective form. The first is drawn from 1,000 autopsies made at the New York Foundling Hospital in about eight and one-half years. It shows 14 cases of acute inflammation of the pericardium occurring at ages varying from six months to three years. The second comprises the cases of acute pericarditis in children observed in the wards of the Presbyterian Hospital during the past four years in a total of about 10,000 patients. The service is, of course, largely composed of adults, but the patients under sixteen years of age number perhaps one-tenth of the total. These tables may be taken to demonstrate that acute pericarditis is not uncommon even in childhood, that it is rarely indeed the primary disease—and that, as we all know, it is a serious affection, and often proves the final catastrophe in severe illness.

Pathology. The pathology of acute pericarditis is so well known that only points of special interest will be mentioned.

The conventional manner of describing several types of pericarditis and denominating them—dry, serous or purulent, or fibrinous pericarditis and pericarditis with effusion—is misleading. Acute pericarditis, we may say, is always marked by swelling of the pericardium and an effusion upon its surface or into the sac. This effusion consists of fibrin, serum and leukocytes in varying proportion. If the serum is scanty and the fibrin abundant, there is little or no fluid in the sac, and the pericardium is coated with a plastic exudate, we speak of the pericarditis as dry or fibrinous. If the serum is more abundant, so that it distends the pericardium, the process is denominated pericarditis with effusion or serofibrinous pericarditis. And if the polynuclear leukocytes in the fluid become abundant and undergo degeneration, we speak of a purulent pericarditis.

For a clear understanding of these conditions it seems very

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desirable to emphasize the well-established fact that these several varieties of pericarditis are but steps in the process of reaction of the pericardium to the assaults of certain noxae, toxins or bacteria. The dry pericarditis of one day may be a pericarditis with effusion the next, and the effusion may eventually prove to be either serous or purulent.

Certain characters of the exudate in acute pericarditis are of practical importance. The amount of fibrin in the exudate varies greatly. In many cases there is sufficient only to coat the pericardium and give it a slightly granular or rough appearance. In other cases the fibrin is so abundant as to cover the heart with a thick plastic layer which may be as much as a centimeter in thickness. The pericardium may therefore be considerably distended and the total width of the area of cardiac dullness greatly increased without the presence of fluid in the sac, and an exploring needle, though it enter the sac, may fail to obtain fluid. In certain cases where pus is present, it may, for like reasons, be obtainable only by an exploring needle of large caliber. The close resemblance in this regard between effusions of the pleura and pericardium in children is very striking.

It is not uncommon in severe cases to observe minute hemorrhage into the pericardium itself, and hemorrhagic exudates are frequently seen. Von Starck* records 4 cases of bloody effusion in rheumatic cases, and our own experience would lead us to believe that the presence of blood is not infrequent in non-tubercular conditions. Chylous effusions are practically unknown in childhood.

The disposition of the effusion with relation to the heart itself is of importance. The conception that the fluid in pericarditis is evenly distributed about the heart is certainly not correct. The fluid tends to accumulate in the lowermost parts of the sac, that is, with the patient supine, in the lower or posterior part. The anterior surface of the heart may therefore be but scantily covered when a considerable quantity of fluid lies behind the heart.

For this reason the effect of the effusion is to distend the pericardial sac downward and backward and there is little or none at all of the assumed floating of the heart itself upward.

It is also clear that the fluid in pericarditis does not quickly or readily change its distribution, and one fails to observe very

* Von Starck, *Jahrbuch für Kinderheilkunde*, 1895, Vol. XL., pp. 70-77.

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prompt response in physical signs to changes of position, such as are possible under the usual conditions. When, however, it is feasible to put the patient in the knee-chest position, an increase in the area of cardiac dullness, not previously observed, may be obtained.

In many cases of pericarditis the myocardium as well is involved and inflammatory or degenerative changes may be demonstrated in it. Beyond doubt the consequent weakening of the cardiac muscle plays a part in causing circulatory failure, and in many cases relaxation of the cardiac chambers is one element in the enlarged area of cardiac dullness.

Associated Lesions. The table of observations from the Foundling Hospital is of interest, showing, as it does, that in every instance but one the recorded pericarditis was accompanied by pneumonia, or pleurisy, or both. The one exception (enterocolitis) is sufficient, however, to support the clinical experience that such association is not inevitable. It is also notable that in these cases the pericardial effusion in 11 out of 14 cases was purulent, and in the remaining 3 sero-fibrinous. In fatal cases we expect the severer lesion, but the frequency of suppurative pericarditis is important.

In early childhood the type of pneumonia seems to be of little importance, lobar and bronchopneumonia both being found together with pericarditis.

The pleural inflammation may be of any grade, fibrinous, serous, or purulent, and it is well known that the two membranes being anatomically of like structure react similarly to any infection and the conditions found in pleura and pericardium in any one case are regularly the same.

Relation to Tuberculosis. In view of the common teaching of the frequency of tuberculous pericarditis, the absence of tuberculosis from the list of conditions associated with or accompanied by pericarditis may excite surprise, but so far as the writer's knowledge goes, the facts are properly stated. In 125 cases of tuberculosis in children under the age of seven years, examined in the postmortem room of the New York Foundling Hospital, tubercles were found in the pericardium of 4 cases, and in the heart muscle in 1, but in none of these cases was there effusion or any other evidence of acute pericarditis.

Clinical experience also corroborates these postmortem findings, for I cannot now recall ever having known a case of peri-

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carditis in a patient under sixteen years of age to be proven by reliable evidence to be tuberculous. It is not, of course, meant to deny the possibility that tuberculosis may produce pericarditis in children, as it is well known to do in adults, but simply to indicate that the tubercle bacillus is very rarely indeed found in this relation.

Bacteriology. Unfortunately in the Foundling Hospital cases no cultures were made, but studies made in the Presbyterian Hospital show that in the pericardial exudates (especially the suppurative) the pneumococcus is most often found, as the frequent association of pneumonia and pleurisy would lead one to expect. Staphylococci or streptococci are occasionally found, but other bacteria (gonococcus, etc.) are very rarely met with.

Bacteria are, of course, most frequently found in purulent exudates. They may, however, be demonstrated in serous or fibrinous effusions. The frequency with which the serous exudates are sterile is noteworthy. Also it must be remembered that the evidence is altogether that these sterile exudates are not tuberculous, but belong for the most part in the rheumatic class.

Poynton and Payne have obtained their "streptococcus rheumaticus" from the pericardial exudate in cases of rheumatic fever, but this we have never been able to do.

Symptomatology. Of the symptoms of acute pericarditis but little will be said, since they are of relatively small importance to the diagnosis.

Dry pericarditis gives rise to very little disturbance, and its onset is marked by the symptoms of the primary condition. There may be discomfort or pain, although in many cases the friction of the inflamed surfaces seems to be scarcely appreciated, and some moderate increase in the pulse or temperature.

With an increasing effusion dyspnea, or orthopnea, cyanosis, and rapid, irregular and intermittent action of the heart develop.

The temperature varies with the nature of the effusion, and may be low or high. In the septic cases there are high fever, pallor of the skin with cyanosis, and frequently profuse sweating. As in nearly all cases the pericarditis is accompanied by acute inflammations of other parts, the heart, the pleura, the lungs, etc., it is quite impossible to satisfactorily determine the relationship of lesions and symptoms.

While in most cases acute pericarditis is a rapid process, quickly evolving its symptoms and physical signs and rapidly pro-

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ducing a crisis in the patient's condition, it is well known that in some instances the onset is insidious. Coutts* has reported a case in which a four-year-old child had been sick for ten weeks before being brought to the hospital, and lived seven weeks longer, with a purulent effusion in the pericardium, which he regarded as primary. During this long period there was nothing in the clinical picture to direct special attention to the pericardium.

Physical Signs. These are the rational signs, first, of a roughening of the serous covering of the heart and then of its distention by accumulating fluid. In dry pericarditis we have the characteristic to and fro rub. When an effusion collects we have the bulging precordium, an increased area of cardiac dullness, enfeebled or lost apex beat and auscultatory sounds, and finally dullness in the cardiohepatic angle, as was years ago demonstrated by Rotch.

Taking the physical signs in conjunction with symptoms of serious illness it would appear that the diagnosis of acute pericarditis in childhood should be readily made. Yet every one knows that the contrary is the fact and that there is no other grave malady so frequently discovered for the first time in the postmortem room.

For the explanation of this situation there are several facts of importance. First of all, that acute pericarditis is a secondary affection, and secondary to some serious primary disease, such as a septic infection, pneumonia, acute rheumatic fever, and the like. The severe symptoms of the primary affection mask the onset of the inflammation of the pericardium. The patient's condition changes for the worse, but the change can readily be explained as the augmentation of the symptoms of the primary disease.

Secondly, the physical signs may be notably modified or obscured by attendant conditions. The pericardial friction sound is justly regarded as a most significant sign of acute pericarditis. Its presence most often first calls attention to the fact that the pericardium is involved and leads to more careful observation. Yet it must be admitted that acute pericarditis can develop and go on to effusion without its being possible, even by the most careful and repeated observation, to detect the characteristic rub. During the last year we had under our care in the Presbyterian Hospital a boy of ten years, whose primary disease was pneumonia, complicated later by a left-sided empyema. The pleura was opened

* Coutts, *British Medical Journal*, 1903, Vol. II., p. 359.

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and drained, and yet the patient did not improve. Day after day most careful study of the heart was made both by myself and an alert house staff. No friction sound was heard at any time, and yet at autopsy a heavy layer of fibrinous deposit was found covering the pericardium. This thick, plastic type of exudate, covering, as it does, the surface of the heart evenly, gives neither the characteristic friction rub nor those of fluid, and is most often overlooked in consequence.

The conditions in the patient to whom reference has just been made well illustrate the second great difficulty, as regards the physical signs of pericarditis, namely, that they are often obscured by the attendant conditions.

It was observed for some days that the limit of cardiac dullness on the right of the sternum was displaced distinctly to the right. This was at first assumed to be due to the empyema, and when it persisted after the opening of the pleura we failed to attribute due importance to it. The presence of the empyema on the left side naturally obscured the limits of cardiac dullness in that direction and left us only the apparent displacement of the cardiac dullness to the right to interpret.

In a similar manner a pleural effusion on the right, a pneumonia, an enlargement of the liver, a marked abdominal distention displacing the liver and diaphragm upwards, or still more commonly the noisy respiration or restlessness of an extremely sick child make observation difficult or misleading.

Recognizing that in this situation any aid to diagnosis will be welcome, I venture to direct attention to a new method of studying old signs with relation to the diagnosis of pericarditis with effusion or serum or pus, the conditions of most vital importance, a method learned from following the careful clinical studies of Dr. Francis P. Kinnicutt.

Increase in the area of absolute cardiac dullness has long been known as one of the most important signs of these conditions. Also that the increased area of dullness had a characteristic form, that of a truncated triangle, the broad base below, the truncated apex above.

The point which the experience of recent years has impressed upon us as of peculiar value is that careful observation will demonstrate, in any case of pericardial effusion large enough to be of consequence, a *progressive* increase in this area, and that such

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progressive increase constitutes the most reliable evidence of a pericardial effusion. (See illustration.)

The detection of progressive increase in the area of cardiac dullness implies the repeated careful examination of the heart and

FIG. 1—Acute Pericarditis, showing the area of cardiac dullness at its maximum, The symmetry of distention is striking. This has been observed in other cases.

the recording of results in some more or less permanent form for later comparison. The outline of cardiac dullness may be plotted on the chest wall, measurements from the mid-line recorded and the findings from day to day compared. But it is immensely more effective if the outline of cardiac dullness can be marked upon the chest wall in some form that will show the changes from day to day. We have done much experimenting in search of a suitable method. The outline may be marked in ordinary ink and renewed from time to time as necessary, but we find that such marks are readily smudged or removed entirely by the nurse's ministrations. Nitrate of silver may be employed, but is so difficult to handle and so likely to cause excoriation, if used too freely, that it has not been found satisfactory. Lately some experiments

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with the ordinary forms of indelible inks have shown that marks made upon the skin with them are more lasting and satisfactory than those made by any other method we have thus far been able to devise.

If, in every case of pneumonia, pleurisy, acute rheumatism and the like in which pericarditis may develop, by some such means the apex beat of the heart and the outlines of the cardiac dullness are marked clearly on the thoracic wall, we shall much more quickly than otherwise note any shifting of the apex beat or its disappearance and any subsequent changes in the area of cardiac dullness. The guiding marks are before our eyes. Furthermore, we shall have much more confidence in them than in measurements recorded in the history, which may easily be wrongly made or wrongly recorded.

FIG. 2—The same patient, showing outlines of cardiac dullness at successive stages of resorption fusing below into area of liver dullness. The outlines shown correspond closely to those observed during the accumulation of the fluid.

With such guides the limits of cardiac dullness may be found to shift in the course of a few hours to a very appreciable extent. It

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is, of course, not the shifting of the limit of dullness on the right or left that is significant, for either alone may be produced by displacement of the heart. It is the outward displacement of dullness on both sides that marks and measures the increasing effusion. Incidentally in following this broadening of the area of dullness we shall appreciate all the more forcibly the dullness in the cardiohepatic angle, and the fact that while the cardiac dullness is increased in all directions the greatest increase is along the base line, *i.e.,* on the diaphragm.

Dilatation of the heart, it may be objected, may give rise to exactly similar enlargement. Granted, theoretically; but practically the objection carries little weight. Dilatation also is an enlargement of either right or left ventricle, rarely indeed simultaneously of both, and therefore has not the symmetrical form of pericardial effusion. In our experience dilatation, unless caused by pericarditis, never occurs in the progressive manner characteristic of the pericardial effusion. Furthermore, the chance of error is removed by observing that the increasing dullness is most marked along the diaphragm and especially in the cardiohepatic angle.

In doubtful cases where the diagnosis is of the utmost importance, it has been found that the area of cardiac dullness in the course of twelve or twenty-four hours will show changes which are decisive. Observation over night or for a day will therefore satisfy one of the presence or absence of pericardial effusion and also give exact important evidence with relation to the question of treatment.

Broadbent and Poynton* both state that such gradual increase in the area of cardiac dullness, associated with dullness in the cardiohepatic angle, may be due to increasing cardiac dilatation, but this the writer has never observed save in the cardiac dilatation resulting from acute pericarditis and accompanied by pericardial effusion.

It should, of course, be understood that the emphasis put upon this particular sign of pericardial effusion is not meant to deny the value of other signs, especially the pericardial friction and the progressive weakening of the apex beat and the heart sounds.

The disappearance of a previously heard pericardial friction may be expected with an increasing effusion, but does not always

* Broadbent, "Heart Disease," fifth edition, 1906, p. 34.
Poynton, "Heart Disease and Thoracic Aneurism," 1907, p. 170.

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follow. Owing to the tendency of the fluid to gravitate to the lowermost part of the pericardium, the upper part may still remain in contact, and the pericardial friction persist even with large effusions. Just where the friction will be heard under these circumstances will depend upon the posture of the patient and the disposition of the fluid.

Signs Over the Left Lower Lobe.—As a collateral sign of pericardial effusion Pins, of Vienna, long ago pointed out evidences of pressure exerted upon the left lower lobe of the lungs and these signs have been repeatedly studied by other observers. Ewart* outlines the area of dullness produced in this manner as bounded by a line drawn outward from the vertebral column at the level of the ninth and tenth ribs and a perpendicular line dropped from the vertebral border of the scapula. Below the dull area reaches the base of the chest and internally is bounded by the vertebral column or it may pass slightly to the right of it. Over the dull area the voice and breath sounds are diminished or absent.

Without doubt in most cases of large pericardial effusion one finds dullness with some modification of the voice and breath sounds, either intensification or diminution, below and internal to the angle of the scapula. Rarely, however, are the signs delimited with the exactness laid down by Ewart's boundaries. The interpretation of these signs is not easy. A partial consolidation of the lung, a pleural effusion, or compression of the lung by the distended pericardium may account for them. The arguments by which Fetterolf and Landis* explain the hydrothorax of cardiac dilatation by pressure upon the pulmonary veins are of interest in this relation. The distended pericardium undoubtedly exerts pressure upon the left bronchus and the lower pulmonary veins, and partially compresses the surrounding lung. But the surrounding lung comprises the upper lobe and the upper part (apex) of the lower lobe, and not the part of the lung corresponding to the area of dullness marked out by Ewart. In brief the compression affects the apex of the lower lobe, while the physical signs are at the base.

Furthermore, we have all, I am sure, followed the gradual evolution of these basal signs of pericardial effusion from day to day, till we have become satisfied of the presence of an effusion in the left pleura, and aspirating it have obtained more or less

* Ewart, see Poynton's work, p. 171.
† Fetterolf and Landis, *Am. Jour. Med. Sc.*, 1909, Vol. CXXXVIII., p. 712.

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fluid. To determine the presence or absence of fluid in the left pleura under these conditions, *i.e.*, with a distended pericardium and the physical signs mentioned at the left base, is most difficult. The extent of the dullness and the diminished voice and breath sounds is probably of most importance. In any case with extensive dullness exploratory puncture of the pleura is called for, in the hope that fluid may be formed and partial relief obtained by its withdrawal.

The studies of Fetterolf and Landis, above spoken of, explain the hydrothorax accompanying cardiac dilatation by the pressure of the enlarged heart upon the pulmonary veins, with consequent stasis in, and serous effusion from, the pulmonary pleura. The same explanation may apply to the physical signs and serous effusion so regularly found at the left base in cases of pericardial distention by fluids.

Neither in the discussion of the physical signs or pericarditis with effusion, nor with reference to the signs at the left base, has mention been made of the information to be had by changes in the position of the patient. It has been claimed that by turning the patient from side to side, or moving him from the recumbent position to the upright, or even from the recumbent attitude to the knee-chest position, valuable information may be obtained with reference to the physical signs and their interpretation. In the great majority of the cases of pericardial effusion, however, the patient is too sick and too uncomfortable to permit of much movement, and efforts to make such comparisons have, in our experience, been of little or no value.

The subject of the diagnosis of pericardial effusion cannot be dismissed without reference to the use of the X-ray. The possibilities of this agency in the diagnosis and study of cardiac lesions in the adult have been fully demonstrated by the remarkable work of Groedel and others. Because of the greater penetrability of the thorax children offer still better opportunities for such study than adults, and yet thus far but little work has been done in this field. Particularly with relation to pericardial effusion in children, it appears that the X-ray may offer invaluable information, displaying as it does not only the limits of the distended pericardial sac, but the position of the heart in the sac itself, so that if aspiration is contemplated the site of puncture may be chosen in confidence that the heart will not be touched.

These possibilities, which to most of us have been a dream,

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have been translated into fact by the pioneer work of Rotch, but a few weeks ago presented to us in his new and most interesting volume, the "Roentgen Ray in Pediatric Practice." In that work are three beautiful plates illustrating the points just stated. The difficulty of obtaining radiographs of severely sick patients has prevented the general use of them in cases of pericarditis, but if such invaluable information can be obtained by this means the difficulties must be overcome, especially as Rotch has apparently demonstrated that they can be.

Aspiration of the Pericardium.—Withdrawal of fluid from the pericardium by a syringe or the aspirator may be undertaken either for diagnostic or therapeutic purposes. Serious objection has of recent years been made to the procedure as so dangerous as to be unjustifiable, and it has been urged that if the pericardium is to be entered it should be done by incision under cocain, so that the operator may see clearly what he is doing. The strength of this position is not to be denied. Attempts to aspirate the pericardium have too often proved aspiraticns of the heart, and death has more than once resulted from the procedure. Various writers speak of having penetrated the heart and drawn blood from it without harm. In the physiologic laboratory blood is drawn from the calf's heart with a canula several times the caliber of an aspirating needle and the canula withdrawn without harm. On two occasions, however, I have seen the heart of an adult punctured and hemorrhage into the pericardium caused with fatal consequences. As it happened, both patients would undoubtedly have died in any event, but the gravity of puncturing the pericardium with an aspirating needle has been deeply impressed upon me. And yet to wholly disregard the procedure because of this danger seems unwise. If exploration of the pericardium is to be undertaken only as an open surgical operation, a limitation will be put upon its use which in the end will work more harm than the older procedure. Moreover, it is possible by adequate care in our observations to make practically certain the diagnosis of pericardial effusion and to practice aspiration with safety. This is especially true in childhood for reasons already pointed out, and in the many aspirations of the pericardium in children which I have seen or done no serious mishap has occurred. Finally, if the Roentgen rays can be employed and such plates as Rotch's obtained, aspiration of the pericardium can be rendered entirely safe.

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Scope of Paracentesis.—A wholesome fear of the use of the aspirating needle in the pericardium has undoubtedly limited its employment. Exploratory puncture must be employed more freely if we are to recognize and treat properly (by drainage) the suppurative conditions which are not uncommon. The nature of the antecedent infection, rheumatic fever, pneumonia, septicemia, cannot be relied upon to determine the nature of the effusions. Suppurative pericarditis has developed in rheumatic conditions and serous effusions been found in pneumonia and septicemia.* Since suppuration demands drainage, safety can be had only by knowing the character of the fluid present in the pericardium, unless we are ready to say that all effusions in the pericardium should be drained.

Secondly, paracentesis may be required for therapeutic purposes. There is a general tendency to rely upon natural processes for the absorption of serous effusions, particularly in rheumatic cases. English writers agree that aspiration in these cases is rarely required, and Poynton says that he has never seen a case of rheumatic pericarditis require such intervention. That view must be extreme. Certainly one sees fatal cases of rheumatic fever in which pericardial effusion is one element in the untoward outcome, and also from time to time one sees rheumatic patients suffering from endo- and pericarditis, and apparently in extremis relieved and helped toward recovery by removal of fluid from the pericardium.

Site of Aspiration.—The fourth or fifth interspace close to the sternum was first selected for this purpose on the theory that the heart was floated upward by the pericardial effusion. Rotch demonstrated the falsity of this conception, and on the basis of his studies recommended reaching the pericardium through the fifth right interspace. Of recent years a point outside the apex and yet within the area of dullness of the distended pericardium has been advocated by West and others. To either of these sites the objection may be urged that to reach the pericardium the overlying pleura (left or right as the case may be) must be penetrated. The great point in favor of one or the other is that they undoubtedly offer less chance of injuring the heart. This fact, supported by theoretical considerations and practical experience, is demonstrated beyond doubt by the radiographs already spoken of. With any considerable effusion into the pericardium there is

* Poynton, "Heart Disease and Thoracic Aneurism," p. 168.

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space on either side of the lower or diaphragmatic border of the heart between the two layers of pericardium occupied only by the effused fluid. Puncture in either of these spaces, so long as it is made in a plane parallel to the median plane of the body, *i.e.*, with the needle parallel to that plane, ought to be entirely safe. Danger arises whenever the needle is given an oblique direction, and the operator is left to try to calculate the position of the point from the line of direction and the length of the needle. Owing to the fact that the distended pericardium is practically always outside the nipple line on the left, puncture outside the apex must be made on the shelving side of the thorax, and it is extremely difficult to avoid a definite inward inclination of the needle with resulting danger of transfixing the apex or the right ventricle of the heart. Furthermore, if the heart is pierced under these conditions the active motion of the affected part (the most active of any part of the heart) tends to enlarge the puncture into a linear wound which is sure to bleed. The two serious results which I have seen in adults were to be explained on these grounds. On the other hand, West* objects to the fifth right space as the most dangerous of all, because of the neighborhood of the thin-walled right auricle. I have safely used both sites, in one instance obtaining the fluid by puncture outside the apex after having failed in the fifth right space, but for the reasons given I have a distinct preference for the latter site.

Prognosis.—The outcome in any case of acute pericarditis in childhood depends, for the most part, (1) upon the primary disease, (2) upon the amount of effusion, and (3) upon the character of the effusion, whether serous or purulent.

The primary disease and the character of the effusion are usually closely connected. In rheumatic infection the effusion is regularly fibrinous or serous. In septicemia, pneumonia, pleurisy and the like it is most often purulent, but may be serous or serofibrinous.

The pure fibrinous or serous exudates regularly resolve with the formation of adhesions. In the rheumatic cases there is a notable tendency to recurrence of the inflammation of the heart and the pericardium, with an ultimate development of chronic endocarditis and chronic adhesive pericarditis. In hospital practice children suffering from these conditions regularly return year after year, and sooner or later surely succumb to cardiac

* West, *London Med. Jour.*, 1908, Vol. XIII., p. 21.

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failure. In private practice results are better, but even under the best conditions it is difficult to prevent recurrences, and the prognosis, so far as concerns active, healthful life, is gloomy.

Suppurative pericarditis in children is fatal in nearly all instances, yet recoveries under appropriate surgical treatment have been recorded. In Eliot's* tabulation of 22 cases of suppurative pericarditis treated by operation, one of the patients was sixteen years of age, and 7 under that age. Of these 1 recovered. This patient was a girl, five years old, who, after a tonsillectomy, developed a left-sided empyema and later suppurative pericarditis. The pleura was first opened and later the pericardium was drained through the empyemic cavity. The child made a prompt recovery. In several of the other cases distinct improvement followed the draining of the pericardium, but the patient died of some further extension of the infection.

The following case illustrative of several of the points made in this paper may here be cited.

J. H., a boy of ten years, was taken sick March 14, 1909, and on admission to the Presbyterian Hospital five days later (the 19th) was found to have a lobar-pneumonia of the left lower lobe. He had the usual high fever, rapid pulse and respiration of that condition. Two days later, in the evening, the patient became very restless and tossed about his bed continually. Examination of the cardiac area showed an extension of the cardiac dullness on both right and left borders of the heart, and a friction sound was heard in the cardiohepatic angle and along the left border. The pulse had risen from 144 to 160, but there was no notable change in the temperature or respiration. The heart sounds were distinct. Twelve hours later (the next morning) the limits of cardiac dullness were further displaced outward, especially on the right, where the line of dullness was outside and above the right nipple, and the cardiohepatic angle was broadly obtuse. The heart sounds were fainter. The friction sounds could still be heard over the upper part of the cardiac area. An aspirating needle inserted in the fifth right space about one inch from the sternal margin withdrew pus. The same morning Dr. Eliot resected a portion of the fifth left costal cartilage, opened the pericardium and inserted a drainage tube. Although little pus was discharged at the time of operation, an abundant discharge occurred shortly afterward. In every way the child's con-

* Eliot, *Annals of Surgery*, 1909, p. 61.

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dition improved for several days, when the right pleura was found to contain pus and was in turn opened and drained. Even under those conditions the child rallied and improved for a time, but died on April 4th, thirteen days after the opening of the pericardium. The pneumococcus was found in both pericardial and pleural exudates.

The apparent hopelessness of these cases should urge us to more careful observation and study, that by early diagnosis adequate treatment may be undertaken as promptly as possible, and the great danger of death, if possible, averted.

The quantity of the exudate, be it serum or pus, has a definite relation to the prognosis. The studies of Haven Emerson* on the effects upon the circulation of fluid (water) injected into the pericardia of dogs have apparently proven that the weak and irregular heart action and falling blood pressure produced by increasing pressure in the pericardial sac is due not so much to the direct effects of the pressure upon the heart itself as to the mechanical cutting off of the return of blood to the heart by pinching or compressing the superior and inferior cava within the pericardium. By reason of this obstruction blood enters the heart with difficulty, the chambers are improperly filled, and the heart exhausts itself in a vain effort to maintain the falling blood pressure.

These studies indicate that whatever the nature of the pericardial effusion, if it be accompanied by low blood pressure and weak, irregular heart action, the pressure within the pericardial sac should be lowered, either by aspiration or drainage.

Treatment.—Rest in bed, careful feeding and nursing are, of course, the essentials. The primary disease must be treated. It is doubtful whether even in the rheumatic cases our remedies have direct influence upon the pericardial inflammation, but the free administration of salicylates has often seemed to check the process. Opium or morphin is of service for the relief of pain and distress. Children, as a rule, object to the application of cold, and the ice-cap seems to have but little effect.

The question of greatest importance is the amount and character of the fluid present. We can estimate the amount from the physical signs and the evidence of interference with the heart action. The character may in many cases be inferred from the

* Personal communication.

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nature of the primary process, but not with surety. With any large effusion it is, therefore, safest to ascertain the character by an exploratory puncture.

Rheumatic effusion will, as a rule, resolve naturally, but certainly removal of the fluid relieves distress and hastens recovery, just as it does in the case of the pleura. With large effusions, aspiration is therefore desirable, and if the conditions are thoroughly studied can be safely done. The removal of even a few ounces of fluid brings definite relief and, in my judgment, earlier recovery.

If the effusion is purulent open drainage is required. For this purpose a portion of the fifth, sixth or even seventh costal cartilage on the left side may be removed, and the drainage tube inserted. Even with such treatment the chance of recovery is small. It will be increased only when we learn to control the primary infections or to make the diagnosis earlier and more promptly institute proper treatment.

TABLE I.

CASES OF ACUTE PERICARDITIS FROM THE AUTOPSY-RECORDS OF THE NEW YORK FOUNDLING HOSPITAL.

No.	Sex	Age	Associated Lesions	Type of Pericarditis
1	Male	9½ mos.	Pleurisy, serofibrinous, and infarctions of lungs	Serofibrinous
2	Male	2½ yrs.	Pleurisy, fibrinopurulent, and lobar pneumonia	" "
3	Male	3¾ yrs.	Empyema	Purulent. Pus, 8 ounces
4	Female	6 mos.	Empyema and bronchopneumonia	"
5	Male	1¾ yrs.	" " " "	"
6	Male	9 mos.	Diphtheria and lobar pneumonia	"
7	Male	1 yr.	Empyema	"
8	Male	2 yrs., 7 mos.	Empyema and lobar pneumonia	"
9	Male	7 mos.	Acute enterocolitis	"
10	Male	9 mos.	Bronchopneumonia	"
11	Female	3 yrs.	Diphtheria, Bronchopneumonia, Empyema	"
12	Male	11 mos.	Bronchopneumonia, fibrinous pleurisy	"
13	Male	6 mos.	" " " "	Serofibrinous
14	Female	10 mos.	" " seropurulent "	Purulent

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TABLE II.

ACUTE PERICARDITIS.—CLINICAL RECORDS OF THE PRESBYTERIAN HOSPITAL, 1906-1910.

No.	Sex	Age	Associated Diseases	Type of Pericarditis	Outcome
1	Male	12 yrs.	Chronic Endocarditis	Serofibrinous. Adh. Peri'dium	D.
2	Male	15 "	Septicemia Empyema	Purulent	R.
3	Female	3½ "	Rheumatic Fever. Endocarditis	Fibrinous.	D.
4	Female	8 "	" " "	"	R.
5	Male	16 "	" " " Lobar Pn.	Serofibrinous	R.
6	Male	13 "	Rheumatic Fever. Endocarditis	" Adh. Peri'dium	R.
7	Male	10 "	" " "	" " "	R.
8	Male	16 "	Endocarditis Chr.	Fibrinous	R.
9	Male	7 "	" "	Serofibrinous " "	R.
10	Female	10 "	" "	Fibrinous " "	R.
11	Male	8 "	" "	Serofibrinous	R.
12	Female	10 "	" " Lobar Pn.	" "	R.
13	Female	4 "	" "	Fibrinous	R.
14	Male	10 "	" "	"	D.
15	Male	7 "	Lobar Pneumonia	Suppuration	D.
16	Male	11 "	" " Empyema	"	D.
17	Male	14 "	" " Pleurisy Fibrinous	Fibrinous	D.
18	Female	7⅔ "	Lobar pneumonia, Pleurisy Fibrinous	Serofibrinous	D.

DISCUSSION.

DR. KOPLIK.—I wish to point out the great difficulty of making a diagnosis of pericarditis in young infants. I have tried it in my service and guided my efforts by postmortem to see if I was correct. In the cases where we thought there was pericardial effusion, there was none; and where we thought there was none there was. The cause of this has been explained by the experimental work of Terrier. The heart lies in such a way that the effusion first begins in the posterior inferior angle of the pericardial sac angle and unless the fluid approaches 200 c.c. the diagnosis cannot be made. In infants the effusion is limited and that is why the diagnosis is possible.

As to the X-ray it is very useful in children but unfortunately, as Dr. Bovaird has said, the cases have to be transferred in order to have an X-ray. In a case recently the X-ray showed very

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beautifully the heart itself and outside the effusion. Without the X-ray the diagnosis was problematical.

As to the area of cardiac dullness I should like to add one point: in children the effusion does not assume that pyramidal shape which it does in the adult; it is more round, because the heart lies horizontal. I would warn against aspiration of the pericardium. In children the exudate is apt to become encapsulated and partitions form. You may not get into the main sac. This happened in a case of mine. The patient was aspirated in a moribund condition; I had warned against it, and the interne did it in my absence. He entered one of these small sacs and through that the ventricle. At postmortem it was shown that he had penetrated the right ventricle.

DR. ABT.—It seems to me the great danger in all these cases of puncture is that it is very difficult to differentiate between a serous or purulent effusion and the so-called acute adhesive pericarditis. Some one has said that 90 per cent. at autopsy show acute adhesive pericarditis; that being true, it would be very difficult to decide when to make a puncture in these cases.

DR. BOVAIRD (closing): I am very much indebted to Dr. Koplik for presenting the reasons for the difficulties that attend the diagnosis of acute pericarditis. I deal with them at some length in my paper but did not bring it out in my presentation of the matter. I tried to make clear my own idea about the extreme care which should be taken in the matter of aspiration. The chief point in the study was my belief that the careful application of clinical methods and the use of the X-ray in these cases will make it possible for us to do safely things that heretofore have been attended with danger.

Acute adhesive pericarditis as is shown in the tables is found frequently at autopsy, but it is my experience that the effusion in these cases shows itself by the same signs as in other instances and that the diagnosis can be made just as if the adhesions were not present. The effusion accumulates in exactly the same way and the pericardial dullness is the same whether there are adhesions or not.

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A CLINICAL STUDY OF 86 CASES OF EMPYEMA IN CHILDREN.

BY FRANCIS HUBER, M.D.,
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In the discussion of empyema in children, the subject must be regarded from two viewpoints—medical and surgical.

Empyema, as observed by the surgeon, differs in many respects from the empyema met with by the general practitioner. The surgeon, as a rule, sees the case late; the family physician, on the other hand, meets the condition early. The cases observed by the surgeon are, so to speak, limited or selected, including, as a rule, only such as have passed the acute stage, and its incidental dangers. The complications have been weathered and the lesion is confined to a purulent collection in one or other pleural cavity.

True, many of the subacute cases coming under surgical care show loss of flesh and strength, are anemic and cachectic, with sallow complexions, and run a more or less septic temperature, with or without diarrhea. Even under such unfavorable conditions the prognosis is favorable.

The practitioner sees a large number, in which various complications exist. Suppurative pleurisy, we must not forget, is only one factor in the infection caused by the pneumococcus or other organism.

Pneumonia, single or bilateral, pericardial and myocardial involvement, meningeal symptoms, single or multiple abscess of lung, gangrene and meningitis are some of the attending conditions met with in the early stages of the disease. The severe constitutional evidences of infection, as convulsions, apathy, even coma, weak pulse, high temperature and typhoid state, add to the dangers in the first week or two. Primary empyema is relatively infrequent. The mortality is high during the early stage because of the various complications referred to above. The clinical course of empyema in infants is short and critical. The postmortem records of Bovaird show that some of the cases die within forty-

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eight hours after invasion, that the greater number of the 69 cases studied by him proved fatal within a week, but few lived longer than two weeks. This phase of the subject must not be lost sight of when the general subject of empyema in childhood is considered. The statistics of the general practitioner, as they include the various types, consequently present a higher mortality.

The affection is much more common in the young, and, unfortunately, more fatal, as the various hospital records show. The prognosis is less favorable, for above-mentioned reasons, in the acute than in those running a subacute course. The outcome depends not only upon the general condition and the surroundings, but also to a great extent upon the presence of complications, the character of the underlying process, the nature of the organism and the degree and intensity of the infection.

Elaborate charts, in the preparation of which I was materially assisted by Drs. S. D. Ehrlich, Harry Cohen, S. Horwitt and S. Ginsberg, giving name, date of admission, and discharge, sex, side involved, age, blood counts, bacteria, urinary examinations, primary operation, secondary operation, recovery, death, complications and remarks, are omitted for lack of space. The main facts are tabulated and presented below.

From January 1, 1908, to January 1, 1909, 35 cases were observed in the children's service at Beth Israel Hospital. As there were 468 children under twelve years of age admitted, the proportion of empyema was about 1 to $13\frac{1}{3}$.

TABLE OF CASES IN 1908 (TABLE NO. I.).

Age.	Male.	Female.	Right.	Left.	Recovery.	Death.	Improved.	Incision.	Resection.	Secondary Resection.
5 months	1		1		1			1		
6 "		2	2			2		2		
8 "	2		2		1	1		2		
9 "		1	1		1				1	
10 "	1			?	1			1		
11 "	1		1			1		1		
1 year	1	1	2		1	1		1	1	
14 months	1		1		1		1	1		
15 "	2			2	1	1		1	1	1
20 "	1	2	1	2	3			3		
21 "		1	1		1			1		1

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Age.	Male.	Female.	Right.	Left.	Recovery.	Death.	Improved.	Incision.	Resection.	Secondary Resection.	
2 years	3	1	3	?	4			4		1	
2½ "	3	1	1	3	3	1		4			
3 "		4	2	2	3			3			1 not operated
5 "	2			2	2			2		1	
5½ "		1		1	1			1		1	
6 "	1		1		1				1		
8 "		1		1	1			1			
10 "	1		1		1			1		1	
	20	15	20	13	27	7	1	30	4	6	1

Two not noted.

FATALITY IN 1908 (TABLE No. II.)

No.	Age.	Male.	Female.	Right.	Left.	Cause of Death.
2	6 months		2	2		Dora N., erysipelas. Dora P., bronchopneumonia.
1	8 "	1		1		Toxemia, two days after admission.
1	11 "	1		1		Gastroenteritis.
1	1 year		1	1		Bronchopneumonia and abscess of lung.
1	15 months	1			1	Erysipelas.
1	2½ years	1			1	Gastroenteritis.
Total 7		4	3	5	2	

From the above we find that the mortality was greater in cases in which the right side was involved.

Admitted during	January, February, March....	10
" "	April, May, June.....	16
" "	July, August, September.....	3
" "	October, November, December	6
	Total	35

As is evident from inspection of Table I 21 were two years of age and under, of which 10 were a year old or less.

One child was admitted on the fifth day of the illness, 1 on the

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eighth day, 1 on the twelfth; 4 had been ill two weeks and the remainder for longer periods.

During the year 1909 the number of cases was greater, the patients were admitted earlier in the course of the disease with a severer type of infection, a most interesting exemplification of the well-known fact of the variation of type in different years.

TABLE OF CASES IN 1909 (TABLE No. III.).

Age.	Male.	Female.	Right.	Left.	Recovery.	Death.	Improved.	Incision.	Resection.	Secondary Resection.	
8 weeks	1			1							refused operation
11 "	1		1			1		1			
4 months	1			1		1		1			
4½ "	1		1			1		1			
5 "	1	1	2		1		1	2			
7 "	3		1	2		3		3			
8 "	3		2	1	1	2		3			
9 "	2		1	1	1	1		2			
10 "	1	1	1	1		2		1			1 puncture and drainage
1 year	1			1	1			1			
14 months		1		1	1			1			
15 "	2		1	1	2			2			
16 "		1		1	1			1			
17 "		1	1				1	1			
18 "		1		1		1		1			
19 "	2	1	3		3			3			
20 "	1	1	1	1		1	not	2			
2 years	4	1	3	2	4	1		5		2	
2½ "	2			2	1	1		2			
3 "	1	2	1	2	2	1		2	1		
3½ "	1		1		1			1		1	
4 "	1	2	3		2	1		3		1	
5½ "	1	3	4		3	1		2	1		1 puncture and drainage
6 "	1		1		1			1			
8 "	1			1	1				1		
9 "	1			1	1			1			
9½ "		1		1	1			1		1	
10 "	1		bilateral			1			1		
Total	34	17	28	22	28	19	3	44	4	5	2

1 bilateral. 1 not operated. 3 improved.

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FATALITY IN 1909 (TABLE NO. IV.).

No.	Age.	Male.	Female.	Right.	Left.	Remarks.
1	11 weeks	1		1		Staphylococcus aureus infection.
1	4 months	1		1		Abscess of lung and bronchopneumonia; died 4 days after operation.
1	4½ "	1		1		Lateral pharyngeal abscess; erysipelas of face; streptococcic infection.
3	7 "	3		1	2	Benny K., bronchopneumonia and abscess of lung. Julius R., died 4 days after operation. Staphylococcus aureus infection. Isie L., bronchopneumonia and sepsis.
2	8 "	2		2		Isie K., death within 24 hours after admission. Isie L., death within 24 hours; pneumonia and sepsis.
1	9 "	1		1		Bronchopneumonia.
2	10 "	1	1	1	1	Rosie P., diphtheria and erysipelas. Max B.
1	18 "		1		1	
1	20 "		1		1	Rosie M., W.B.C. 40,000; poly. 80 per cent.: streptococcic infection; death 9 days after operation.
1	2 years	1		1		Bronchopneumonia, enterocolitis, septic purpura.
1	2½ "	1			1	Bronchopneumonia and pericarditis.
1	3 "	1		1		Gangrene of middle lobe.
1	4 "		1	1		Bronchopneumonia, erysipelas, acute suppurative pericarditis.
1	5½ "		1	1		General pneumococcic infection.
1	10 "	1		bilateral		Pyopericarditis.
Total 19		14	5	12	6	

Admissions, 1909, according to seasons, as follows:—

January, February, March	10 cases
April, May, June	17 "
July, August, September	9 "
October, November, December	15 "
Total	51 "

Duration of illness: 25 were sick two weeks or less as follows:—

Ill 2 weeks	11 cases
Less than 2 weeks	14 "

1 admitted third day of illness; 2 on the fourth day, 1 on fifth day, 1 on sixth day, 3 on seventh day, 2 on eighth day, 2 on tenth day, 1 on twelfth day and 1 on thirteenth day.

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Comparing 1908 and 1909 we find that in 1908, of 468 children under twelve years admitted there were 35 empyemas, a proportion of about 1 to 13⅓. In 1909, of 499 admitted, 51 were empyemas, practically 1 to 10.

In 1908, 20 males and 15 females; right, 20; left, 13.*

In 1909, 34 males and 17 females; right, 28; left, 22.

In 1908, 21 were two years of age or under, of which number 10 were one year old or less.

In 1909, 33 were two years of age or under, of which 17 were one year or less (one-third of the total number).

In 1908, 7 cases had been ill two weeks or less. In 1909 the number was 25—a marked contrast.

In 1908 we lost 7, 4 males and 3 females. In 1909 the mortality was 19, 14 males and 5 females. In both years the mortality was greater in males.

Right-sided cases gave a larger mortality—thus in 1908 it was 5 right and 2 left; in 1909, 12 right and 6 left.

In institutions in which strict isolation cannot be carried out, epidemics of infectious diseases will take place. Thus in the early part of 1908 an epidemic of measles broke out and 6 of our empyemas were affected. In addition, they showed Klebs-Loeffler bacilli without clinical manifestations, in cultures taken from the nose and throat. Fortunately there was no fatality, a circumstance which I attribute to the devotion and attention given to the patients by Dr. W. G. Wulfahrt, in charge of the isolation ward at the time.

Cases of more than passing interest are briefly referred to below:—

A case of empyema and purpura, treated in the service, has already been reported in the ARCHIVES OF PEDIATRICS, November, 1908.

In the case of Jos. B., six years of age, a rib resection was followed twenty-four hours later by symptoms closely resembling peritonitis. There was marked depression, prostration with copious vomiting, decided pain with tenderness and pronounced distension of the upper half of the abdomen. As the vomitus was copious, yellowish at first and later on greenish, with a foul odor and larger in amount than ingested, Dr. H. M. Silver, who kindly saw the case with me, suggested an acute dilatation of the stomach. Lavage, repeated several times, relieved the distressing symptoms in a short time. Further course uneventful.

* In 2 cases, the history failed to state the side involved.

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Physical Signs.—It is not deemed necessary in this discussion to dwell upon the physical signs in detail. Flatness, increased resistance to the percussing finger, absent or diminished breath sounds or distant breathing* (frequently bronchial in type), are characteristic, particularly with a displaced heart.† The displacement is greater in left-sided effusions, because of the normal position of the heart to the left.

Edith S., admitted April 27, 1910. Operated April 28th. Fifteen months old. Right empyema, rib resection; ill five weeks. Blood count, 22,000; 66 per cent. polynuclears. Recovery.

Displacement of the heart is a most important sign in the differential diagnosis. The position of the visible impulse is to be noted, whether to the right or left of the sternum; if behind the bone it cannot be appreciated. At times, however, the impulse may be evident just below the ensiform cartilage. Palpation may aid when the impulse cannot be made out by inspection. In many instances auscultation must be resorted to in order to locate the apex beat.

* The importance of a recognition of "the sense of resistance in diagnosis" is emphasized in an editorial in the *Medical Record,* June 4, 1910, which has appeared since the writing of this article.

† The breathing may be loud and bronchial over a purulent effusion. The "board-like feel" and displaced heart will aid in a correct interpretation of the findings.

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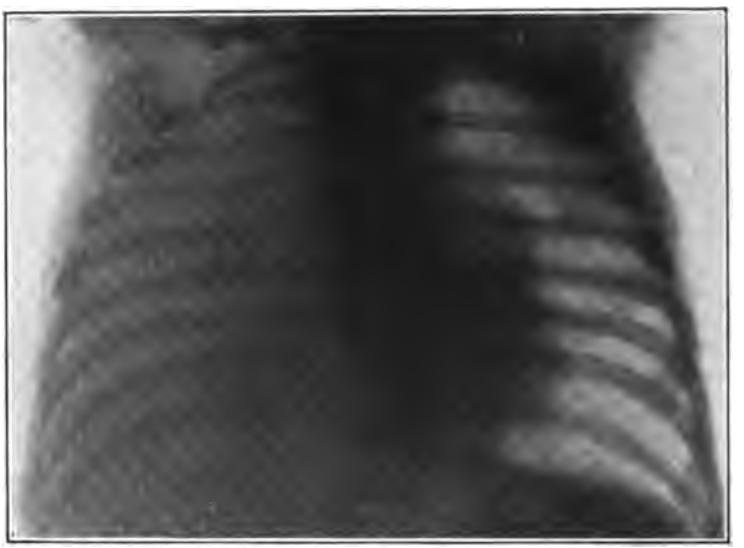
Fluoroscopic examinations give positive and valuable information. With the aid of the screen we frequently find unsuspected pulmonary consolidation in addition to the effusion.

The books tell us that the affected side is more prominent with wider intercostal spaces. In cases of large effusions and of long standing, this may be true. In the greater number the intercostal spaces, on the contrary, are narrowed and the side is retracted.



EMPYEMA (LEFT).

Morris P., fifteen months, admitted February 12, 1908, had measles four months ago, followed by pulmonary complication and cough for two months. Lost considerable flesh; became very pale.
Twelve days ago ran up temperature and began to cough again; was quite prostrated; pneumonia diagnosed; improved in five or six days, to again relapse. Taken to dispensary. Empyema diagnosed.
Rachitic chest. Left side posteriorly, from angle of scapula to base, marked dullness, diminished breathing, distant bronchial fremitus and voice. Above breathing bronchial, numerous moist râles. Anteriorly, dullness and diminished respiratory sounds.
February 12th, white blood corpuscles, 14,400; polynuclears, 68 per cent.
February 13th, exploratory puncture negative.
February 17th, exploratory puncture negative.
February 19th, nose and throat culture positive. Isolated.
February 23d, white blood corpuscles, 20,000; polynuclears, 78 per cent.
February 27th, rib resected in axilla.

Diminished negative pressure within the thorax may account for the lessened normal depression of the intercostal spaces. Because of the pain, the affected side is favored. The approximation of the ribs in the early stages may be attributed to the spasm of the intercostals, analagous to the reflex muscular contraction observed in joint disease. At the same time a marked resistance to pres-

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sure is met with in the interspaces. Narrowing of the intercostal spaces and scoliosis (with the convexity toward the unaffected side) have been observed even with large effusions three to four weeks from the onset of the trouble. Compensatory emphysema of the other lung further tends to accentuate the disproportion. The X-ray studies emphasize these points in a striking manner.

The absorption of the liquid portion of the exudate in chronic cases gives rise to a retraction of the side involved, with more or less deformity.

Though numerous bacteriologic studies have been made, we are not in a position at present to separate empyema into sharply-defined groups corresponding to the bacterial findings. In 36 cases in our series pneumococci occurred nineteen times, staphylococci eight and streptococci nine. Other writers give a higher percentage of pneumococcus infection; no doubt our own tables would have presented a larger number if the results of the examinations had been recorded. The organism shows a marked tendency to die out. Thus, in a case recently observed, no organism was found at the time of operation, about the twentieth day of the disease. In not a single instance did we find tubercle bacilli.

Pyocyaneus contaminations occurred rather frequently, always as a secondary ward infection. Mixed infections were only noticed with putrid exudates. The relation of the pleura to the deeper portions of the respiratory tract (where mixed infections are less frequent) is advanced to explain the limited number or, rather, varieties of organisms giving rise to empyema. Pneumococci were usually discovered in those cases in which large fibrin masses existed in the turbid or purulent fluid.

Text-books speak of primary and secondary types of empyema; in practice, however, it is not always easy or possible to assign cases under one or other category. Pleurisy, it must be remembered, is nearly always secondary to disease in neighboring organs, particularly the lungs. Primary empyema is said to be more common in children. The question is not easily settled. The presence of fluid may obscure underlying pulmonary conditions, while the absence of sputum adds to the difficulty. Fluoroscopic examination not infrequently reveals areas of pulmonary consolidation not detected on a physical examination.

Bovaird, who has carefully studied 101 fatal cases at the New York Foundling Asylum, writes: "The number of cases in which the lung presented no lesion, but compression (35 per cent.),

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is striking. Nor will it do to explain away this high percentage on the hypothesis that a pulmonary lesion had been present before the empyema, but had resolved before death. The great majority of these cases have been so acute and fatal within such brief periods (even two to three days) that we must believe that the conditions found at autopsy were substantially those present during life."

In our experience primary empyema is not nearly so common, probably not more frequent than 1 in 10. A plausible explanation of this discrepancy is that our cases do not include the very early type (two, three and four day cases).

Secondary empyema may follow pneumonia (either catarrhal or lobar), pulmonary tuberculosis (less frequent in children), and abscess or gangrene of the lung. In none of our cases was there a history of trauma. In a few suppurative otitis was present. In 5 erysipelas attended or preceded the trouble. (One of these with vaccination.)

Gangrene leads to putrid exudates. In 3 cases under our observation the odor was not present at the time of operation. In fact, it was not observed until several days had elapsed.

Sarah M., aged fourteen months, admitted May 30, 1909, and Dora M., four and one-half years, admitted April 7, 1908, both presumably with ordinary empyema, a few days after operation developed a foul odor from the wound. When careful irrigation was resorted to a paroxysmal cough followed, and when a weak peroxide solution was employed a frothy fluid was coughed out, showing a communication with a bronchus. The younger child recovered in about six weeks. The older patient was left with a pulmonary fistula, which, after a number of months, finally closed.

Secondary types have been observed after scarlet fever, measles, whooping-cough, multiple furunculosis and marasmus. In one case, after a severe burn, observed a few years ago, a double empyema developed subsequently.

Encysted empyema is not infrequent. The maintenance of one position during the development of the process influences the location of the effusion. If the patient be kept in a dorsal decubitus, the fluid is usually confined to the lateral and posterior regions of the affected pleura; if kept lying on the side a corresponding limitation may be observed.

In the 101 fatal cases studied by Bovaird, he found that, "as a rule, a part, not the whole, of the pleural sac is involved in any

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given case. Usually it is the pleura covering the lower lobe that is inflamed."

The cases reported below illustrate some of the conditions referred to above:—

Nathan S., four and one-half months old, admitted October 7, 1909, died four days after operation. A small abscess of the lower lobe of the right lung was found in addition to an extensive pneumonia.

EMPYEMA (RIGHT) LOCALIZED.

Elsie S., twenty-one months, admitted February 13, 1908. Five weeks ago, fever, rapid pulse and cough. Temperature high for about twelve days, when it dropped to normal by crisis.
Temperature remained normal for a short time; then began to fluctuate daily between 100 and 105° F. No great dyspnea, cyanosis or prostration during the disease. Pleuropneumonia (right).
February 13th, white blood corpuscles, 21,000; polynuclears, 81 per cent.
February 27th, exploratory puncture negative posteriorly.
February 28th, exploratory puncture positive in axilla.
February 29th, operated (resection). Recovery.

The case of Benny K., seven months of age, of pyopneumothorax, is merely referred to, as a more detailed report has appeared in the ARCHIVES OF PEDIATRICS, April 10, 1910.

Yetta K., five and one-half years old, admitted September 27, 1910, with a pyopneumothorax probably following an exploratory puncture before admission, is briefly discussed in the same article.

Annie A., nine years old, admitted on the eighth day in a comatose state, delirious, restless, weak pulse, dry, brown, heavily-

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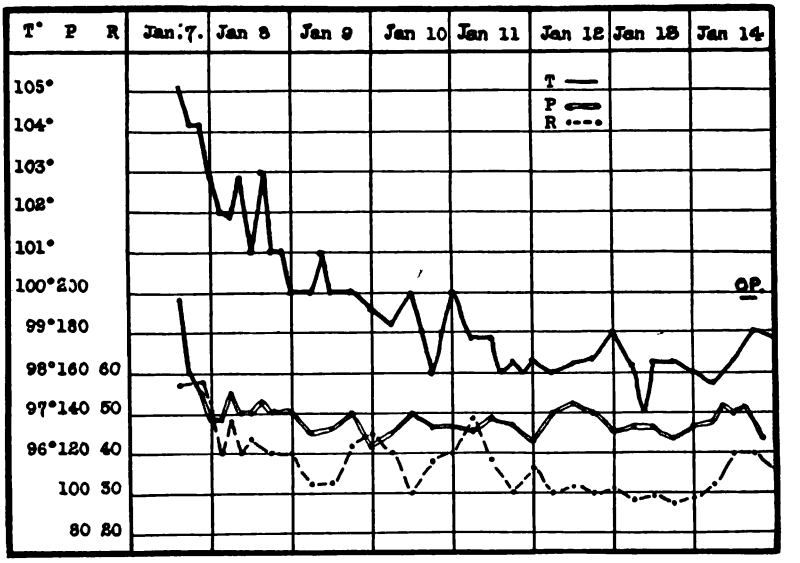
coated tongue and sordes on the teeth and gums. Temperature, 104° F.

As the outcome was not promising, the chest was aspirated and irrigated with a weak solution of formalin; alcoholic and other stimulation was resorted to. A few days later, simple incision was done, followed by a rib resection subsequently. Recovery.

Not less interesting and instructive was the case of Archie S., nine years of age. The empyema developed during the course of



EMPYEMA POSTPNEUMONIA (LEFT SIDE).

Isaac Z., fifteen months old, admitted December 29, 1909. December 24th, sudden onset with fever, cough and dyspnea. On admission: Left lower lobe dullness, bronchial breathing, râles and increased fremitus. Course, see "T" Chart. Decline gradual to January 3d; process now extended upwards (irregular "T" to January 8th). Though fever less, child did not rally. January 15th, extensive effusion (Skoda resonance obliterated). Exploratory puncture negative posteriorly, pus found laterally. Pneumococci present.
January 18th, incision and drainage (large amount of pus and fibrin escaped). Recovery.
December 29, 1909, white blood corpuscles, 19,600; polynuclears, 88 per cent.
January 8, 1910, white blood corpuscles, 19,000; polynuclears, 67 per cent.
January 16, 1910, white blood corpuscles, 24,000; polynuclears, 78 per cent.

measles. His condition was desperate at the time of admission. He was aspirated, and a few days later a thoracotomy was done. Further progress favorable.

In the following case there had been a coincident meningitis:—

Alex R., two years old, admitted April 2, 1909, in coma; pupils dilated and not responsive; head would fall in any direction (due to loss of muscular tone). The condition resembled that of a

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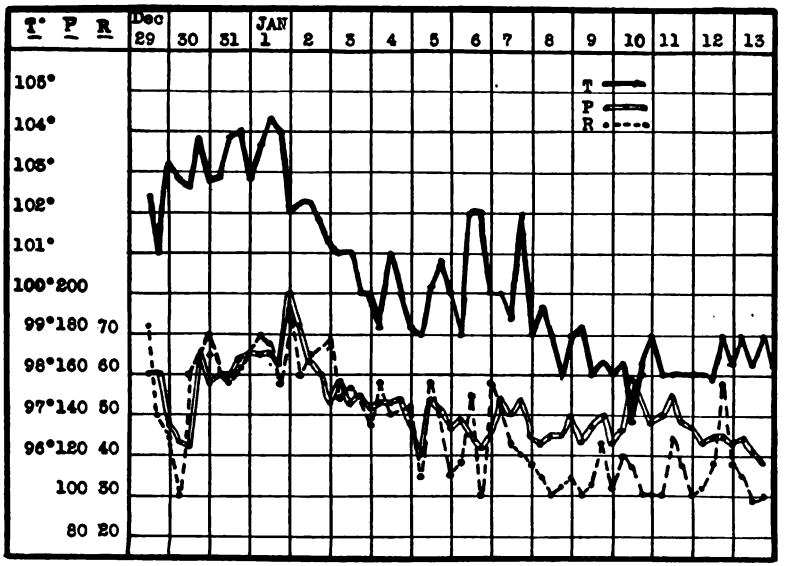
chronic hydrocephalus. Duration of trouble, about four weeks. The chest was incised; condition desperate for weeks. A lateral pharyngeal abscess subsequently developed and was opened. Was later sent to the Seaside Sanitarium. Under the more favorable conditions the patient gradually regained strength, the mental state improved, and, on his return to the city in the fall, he was able to walk, talk and recognize objects.

The symptoms in the acute cases are not distinctive. An apparently frank pneumonia terminates by crisis or lysis; the child,



PRIMARY EMPYEMA (RIGHT SIDE).

Lena S., two-and-one-half years old, admitted January 7, 1910, came in on the eighth day of the disease in a desperate condition; 200 c.c. clear serous fluid withdrawn to relieve the dyspnea. Improvement. January 11th, exploratory puncture revealed yellow turbid fluid. January 12th, pneumococci found in fluid with 90 per cent. polynuclear cells.
January 8th, white blood corpuscles, 9,600; polynuclears, 70 per cent.
January 10th, white blood corpuscles, 28,000; polynuclears, 72 per cent.
January 14th, white blood corpuscles, 22,000; polynuclears, 74 per cent.
January 14th, operation (simple incision and drainage). Recovery.

however, does not rally; upon physical examination, a more or less extensive effusion is discovered. The temperature, on the other hand, may continue and assume an irregular type. In some cases, though the onset may have been severe, the subsequent course after the eighth or tenth day or later is mild. Little or no dyspnea is noticed as long as the child is not excited; the condition is remarkable, in view of the fact that the chest may be full of pus. As a practical illustration, the above *charts* are introduced.

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In the further course there is loss of strength and flesh, with more or less prostration. The patients become anemic- and cachectic-looking. The superficial veins over the affected side are enlarged and prominent. Respiration and pulse are accelerated.

Clubbing of the fingers is generally a late manifestation. The skin may be shining and smooth from obliteration of the normal furrows and depressions. Edema of the skin is not very common, and is usually a late manifestation. On the other hand, edema of the affected side may occur early in infants. The displacement of the heart and liver is found to be relatively greater in purulent cases, probably due, as Senator suggests, to the greater weight of the purulent effusion.

Relapse or recurrence of an empyema is not of frequent occurrence. Incomplete absorption or evacuation of the pus and disintegrated fibrin may be followed by symptoms which lead us to suspect a return of the fluid.

Insufficient drainage or the too early removal of the drain, encapsulation or undiscovered pockets and the imperfect expansion of the lung may lead to a further accumulation of pus. In a few instances, local necrosis of the ribs leads to a recurrence.

Pryor (*New York Medical Journal,* December 21, 1907) writes: "The fact that empyema may recur years after recovery has not received the attention it deserves. It seems to be a very rare affection." The subject has been discussed by the writer in THE ARCHIVES OF PEDIATRICS, July, 1908, and additional cases were reported.

Perforation of the lung, thoracic walls, diaphragm, or esophagus, did not occur in any of our cases. Pericardial and mediastinal infections were fairly common, though frequently overlooked. Metastatic abscesses elsewhere were seen now and then. Pockets of pus may keep up the temperature and lead to a fatal termination.

Amyloid changes were not observed, nor did we have a single case of pulsating empyema.

The accidents which may follow exploratory puncture or aspiration have been fully considered in a former article (ARCHIVES OF PEDIATRICS, March and April, 1910), and need not concern us here.

In the fatal cases, which we have been able to study by "inspection of the wound" or exploratory punctures of the pericardium and spinal canal, other organs were involved in every in-

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stance. We have found extensive lobar or bronchopneumonia, single or multiple abscesses of the affected side, and sometimes of the other lung as well, pericardial involvement or turbid fluid in the spinal canal. Parenchymatous degeneration of the liver, kidneys and heart were frequent findings.

Erysipelas, starting in the wound and spreading rapidly, contributed to a fatal end in 3 cases. In 1 case gangrene of the middle lobe was found at autopsy. The history in brief is as follows: Max T., aged three years, admitted April 14, 1909. Measles seven weeks before. Three weeks later pneumonia, with apparent recovery on the twelfth day. After a few days again taken ill and then sent to hospital with diagnosis of empyema. Thoracotomy same day, sixteenth, rib resected. On the eighteenth, four days after operation, first noticed the gangrenous odor to the discharge. Death April 21st.

Careful urinary examinations were made in our cases. At times traces of albumin and a few hyalin casts were present, findings explained by the fever, toxemia or, now and then, the result of stasis.

In a few instances, in patients with diphtheria or erysipelas, a complicating nephritis was present.

Blood studies made in the majority of cases, though of scientific interest, did not assist us materially from the clinical standpoint. They were of but little value in diagnosis or prognosis. The white cells ranged from 14,000, with 60 per cent. poly, to 40,000 and 80 per cent. poly.

In the case of Ber. K., fifteen months old, ill three weeks, there were 20,000 white cells, with 52 per cent. poly (recovery). Case of Beckie G., two years, 28,000 white cells; poly, 92 per cent. (recovery). Max B., ten months old, ill two weeks, had a count of white cells of 40,000, with 62 per cent. poly. In his case there was a general erysipelas, with meningeal involvement, and the spinal fluid contained streptococci. The case terminated fatally.

The diagnosis of effusion having been made, *exploratory puncture* is indicated to establish the nature of the fluid. A negative puncture is not conclusive; the pus may be thick or the needle be too small, or it may have penetrated some of the large fibrin masses so commonly disclosed at the time of operation.

Decision as to the nature of the turbid fluid, whether pus or not, is difficult at times. Many of the cases are on the border line between the purulent and serofibrinous types.

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Furthermore, the tendency of pus to sediment in the chest must be remembered. Not infrequently a specimen from the upper layers may be turbid, while pus is found lower down.

In encysted cases, or when the signs are atypical, radioscopy is of material service.

Treatment.—The medical treatment varies with the individual case. The indications must be met as they arise. As breathing is more or less impeded by the effusion, particular attention ought to be paid to the gastrointestinal tract. Tympanites in particular must be prevented by a judicious diet, colonic flushings with a low pressure or the insertion of a catheter into the rectum at regular intervals. To quote from a previous paper, read before the Academy of Medicine, May 19, 1887, I would say no reliance can be placed upon measures intended to produce absorption of the purulent products. Our therapeutic resources enable us to combat special symptoms and improve the general condition with the aid of proper food and stimulants of various kinds. Good hygienic surroundings are essential. During the active inflammatory stage, symptomatic treatment is indicated. Caffein, strych., am. carb., liq. am. anisat., etc., are of value.

Border-line cases, in which the effusion is turbid and slightly purulent, and cases of serofibrinous effusions, are benefited by medical measures; pus cases are distinctively surgical. Exceptionally, aspirations, perhaps repeated, may result in a cure.

Aspiration, as a tentative measure, has its indication, and is a valuable aid in tiding cases over danger points when the amount of fluid is large, the general condition bad or the constitutional symptoms severe.

In perhaps 1 in 10 cases admitted to our hospital service aspiration of from 4 to 6 ounces is indicated because of severe dyspnea, cyanosis or rapidly developing cardiac weakness, with or without pulmonary edema or excessive amount of fluid with great displacement of the heart. The danger is greater when the left side is involved.

Thoracentesis is a temporary expedient; more radical measures are subsequently required.

From the surgical standpoint, empyema may be regarded as an abscess, with partly rigid and partly flaccid walls. Thorough drainage having been secured, (*a*) the cure is accomplished by the falling in of the chest walls; (*b*) resumption of the proper posi-

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tion of the organs or structures displaced by the fluid, and (*c*) expansion of the compressed lung. The last factor is of the utmost importance; unless the lung is in a condition to expand, cure will not result until the empyemic cavity is obliterated by extensive resection of ribs and the thoracic pleura.

As to the time of operation—in the absence of sepsis, diarrhea or other unfavorable symptoms—I am in accord with the view of those authorities who prefer to delay radical operation until the acute inflammatory process has subsided and the attending complications are under control. To add a traumatic insult at the height of an acute infection invites additional dangers and may tend to lessen the patients' chances.

In a few patients, in whom the condition was so desperate at the time of admission and who would not have withstood the shock of an operation of any gravity, puncture with trocar and cannula and drainage with rubber catheter of small caliber was employed for a few days until more radical means could be resorted to. Primary rib resections were made in 8 cases, 4 in the series of 1908 and 4 in 1909.

With the above-mentioned exceptions, the rest of the cases were operated upon by simple incision and drainage; a double drain, with strips of balsam of Peru gauze on either side, was inserted into the pleural cavity and the usual dressings applied. The dressings were changed whenever pus appeared through the compound pad.

As the greater number of our cases were associated with pneumonia, the temperature after operation would continue until the pulmonary condition improved. Otitic complications not infrequently gave rise to more or less irregular fever. Pyocyaneus infection occurred now and then as a secondary process, and would give rise to an irregular type of fever persisting for ten days to two weeks. In from six weeks to two months the greater part of the cavity would be obliterated and a sinus only remain. The latter would generally close within a few weeks or month. In 10 cases secondary rib resections were required. At the operation, bony bridges were usually found, similar to the photographs on page 192. Cure readily followed.

Anesthesia was rarely employed. In simple incision, with drainage, it is not necessary; in the rib resections, particularly secondary resections, anesthol was the anesthetic of choice.

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ELSBERG'S POSITION.

For years our cases have been operated upon lying flat on the abdomen, the incision made one or two interspaces below the angle of the scapula, simply because the position was more convenient, and as anesthesia was but rarely employed, the little patients could be restrained better.

The healthy side is not affected in any way or manner, and breathing is not further embarrassed. Cyanosis or other threatening symptoms frequently arise when the unaffected lung is compromised in its movements.

"The experiments of Elsberg demonstrate that the position has manifest advantages. It is a very simple method of safeguarding our patients a little more in intrathoracic operations" (*Journal Experimental Medicine,* 1909, Vol. XI., No. 3).

With the patient flat on the stomach, the head can be turned to one side or supported beyond the end of the operating-table and the anesthesia given without trouble.

The plan suggested by J. B. Murphy, of Chicago (formalin-glycerin injections), has been employed on several occasions. The solution used is a 2 per cent. solution of formalin in glycerin, made at least twenty-four hours before using, in order to insure complete solution of the formalin. After aspirating the greater portion of the pus or fluid, inject 20 to 200 c.c. of the solution, and repeat it every third day until the fluid aspirated loses its purulent character. The quantity injected may be doubled at each injection, until 400 c.c. is reached, and the interval may be lengthened.

The results have not been satisfactory thus far. It is possible that we may not be familiar with the technique.

About three-quarters of our cases are pneumococci infections, the pus containing large flakes and masses of fibrin. It is consequently difficult to draw off a great deal of fluid, for the fibrin is apt to clog the needle or cannula. When, under these circumstances, the solution is injected, it may possibly be too dilute to accomplish the desired effect. The fluid withdrawn at the subsequent tappings was found to be decidedly less purulent, due not so much to a change in the character of the inflammatory process, but rather to a deposition of pus cells. The fibrin masses also would become more brittle and tend to disintegrate. In a few cases there would be a good deal of leakage into the tissues of the thoracic walls, giving rise to a burning pain and decided

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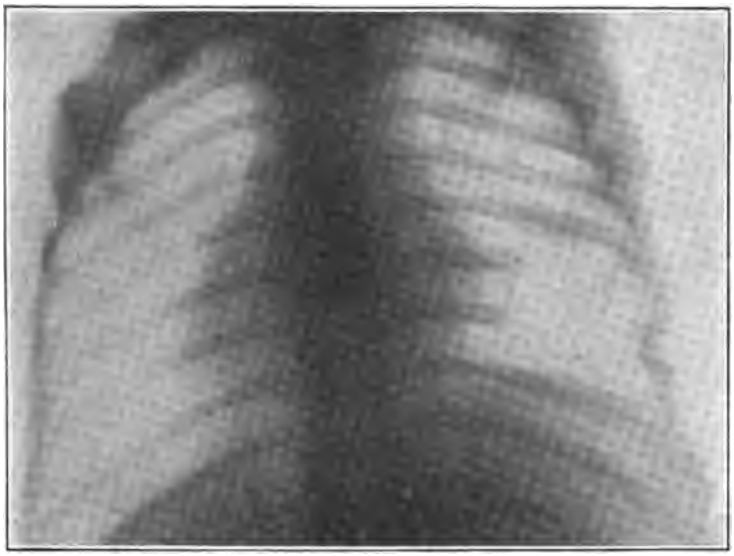
tumefaction. The former would disappear in a few hours, the swelling persisting for days; when such tissues were incised, they would cut like brawn, bleeding but little. On two occasions there was a steady flow from the puncture, necessitating immediate incision and drainage.



X-ray photo of Able W., taken by Dr. I. S. Hirsch, June 16, 1909, a secondary rib resection having been performed June 7, 1909. (Specimen Fig. 3.)

X-ray photo of Able W., taken by Dr. I. S. Hirsch, April 12, 1910, showing restoration of bone, the last operation having been performed June 7, 1909.

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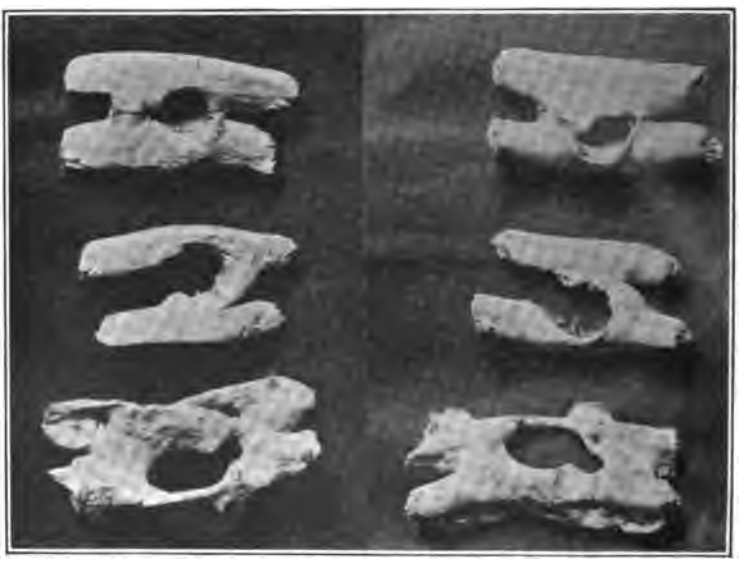
A persisting sinus may be due to a variety of causes; these will vary with the individual case. Imperfect expansion of the lung, due to poor muscular development, an excess of granulation tissue in the deeper portions of the wound, necrotic bone or the existence of bony bridges (as shown in photo), preventing a falling in of the ribs, are some of the factors leading to the production of a sinus in cases of simple incision with drainage. Thickening of the pleura, interfering with pulmonary expansion, is a frequent



ANTERIOR VIEW. POSTERIOR SURFACE.

Resected ribs, showing bony bridges and new bone formation. (Reduced one-half. Photos by Dr. I. S. Hirsch.)
FIG. I. (Upper Specimen)—Charley R., five years of age. Thoracotomy, April 20, 1908. Resection, September 8, 1908.
FIG. II. (Middle Specimen)—Becky J., fifteen months old. Thoracotomy, October 12, 1908. Rib resection, January 3, 1909.
FIG. III. (Lower Specimen. New bone formation)—Able W., four years old. Thoracotomy, August 31, 1907. Resection, November 12, 1907. Secondary resection, June 7, 1909.

cause in chronic cases. Following resection of one or more ribs, necrotic pieces of bone, granulations in the wound and a thickened pleura may be the cause of a sinus not healing. Excessive probing and too long retention of the drain must be avoided.

After operations, patients are allowed out of bed as soon as the condition will permit, in order to improve their general health and muscular tone. Nasal obstruction is relieved by removal of

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adenoids and tonsils, to secure better and deeper breathing. Massage of the chest and proper exercise, to favor expansion of the lung, is advised. The children are encouraged to blow soap-bubbles, or force colored fluids from one bottle to another, as advocated by Dr. W. B. James.

Beck's method of treating the sinus was disappointing to our experience, though the technique advised was carried out in a number of appropriate cases. Apparent cures were followed out by renewed suppuration, with discharge of pus and bismuth vaseline.

Examinations of many of our patients, months and years after recovery, show that, aside from the scar and new bone formation, there are few or no evidences of untoward sequelæ in cases operated upon early, that is, within a few months from the beginning of the illness. Pulmonary expansion was good, breathing and voice unchanged, no retraction or deformity of the chest noticed. X-ray studies of some of the children, by Dr. I. S. Hirsh, confirm the above statements. In chronic cases, with thickened pleura and interstitial changes in the lung, there may be only partial re-expansion with considerable thoracic deformity. West reports 2 cases of serofibrinous pleurisy, in which the lung was capable of expansion after eighteen months. (*Lancet,* March 25, 1905.)

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A CASE OF MEDIASTINAL CYST PRODUCING COMPRESSION OF THE TRACHEA, ENDING FATALLY IN AN INFANT OF NINE MONTHS.

BY A. D. BLACKADER, M.D.,
Professor of Diseases of Children, McGill University,
AND
DAVID J. EVANS, M.D.,
Lecturer on Obstetrics, McGill University.

The following case, which presented symptoms closely resembling those reputed to arise from compression of the trachea by the thymus gland, is sufficiently interesting to warrant our placing the details of it before this Society. For the privilege of seeing it and for the history of the case Dr. Blackader was indebted to Dr. David J. Evans, of Montreal, the physician in charge.

P. S., male, was born July 28, 1909, after a rapid and comparatively easy labor. The child was nursed by its mother until he was over six months of age, when weaning took place. At this time its weight and general condition were excellent and no difficulty either with respiration or deglutition was noticeable. When seven months old the infant contracted German measles, of which there were several cases in the household. During the attack no special symptoms were noted, but after it passed off bronchitis, with frequent loose movements from the bowels, set in, and brought on a noticeable interference with general nutrition. While this condition persisted, the temperature was only slightly elevated, but at times the respirations were of such a peculiar, croupy type that Dr. Birkett, a laryngologist, was asked to examine the infant. He reported that he could see no condition about the larynx to account for the noisy respiration.

While the bronchitis persisted, the child was confined absolutely to two rooms in the house, in both of which care was taken to maintain an even temperature and good, effectual ventilation. A few weeks later indications of rachitis developed, with sweating of the head and tenderness and slight enlargement of the costochondral articulations.

Toward the end of March the child improved again, took nourishment freely and gained in weight. Throughout all this period the breathing, as a rule, was quiet and natural; but occasionally, without apparent cause, sudden dyspneic attacks oc-

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curred, and were associated with moist râles at the base of both lungs.

On the first of April the child became suddenly worse; the mother said it had caught a fresh cold, which she attributed to having taken greater liberty in moving it from room to room in the house. The breathing now became labored and difficult; respirations rose in frequency to between 40 and 50 per minute, and expiratory stridor was distinctly evident. A physical examination showed a hyperresonant condition over the whole chest; but air seemed to enter the air spaces freely. No enlargement of the heart was apparent. At the upper part of the chest anteriorly there was an area of definite dullness, extending both right and left of the sternum and downward to the middle of the second costal cartilage. There was no pyrexia. The child took its nourishment fairly well, and at times played with its toys.

During the next two days it remained in about the same condition, though sleep was frequently disturbed by attacks of coughing and of dyspnea. On the evening of the third Dr. Evans regarded the condition as distinctly improved. Early on the following morning, however, the dyspneic attacks became more severe and the respirations increased to between 60 and 70 per minute.

During these dyspneic attacks the infant had an appearance as if struggling for air. There was a slight bluish tinge about mouth and nose, but no evidence of cyanosis in the fingers or toes. Each attack lasted a few minutes, and ended by the child expelling a quantity of flatus. After this it gradually returned to a quieter condition, looking pale and exhausted, while the head was bedewed with moisture.

In the afternoon, the attacks came on with increasing frequency and lasted four or five minutes at a time. In the intervals the child remained in a somewhat torpid condition and refused to swallow anything, struggling violently if any fluid was put in his mouth.

At nine o'clock that evening the infant was seen by Dr. Bell, Dr. Birkett and Dr. Blackader. A physical examination revealed an area of definite dullness extending 3 cm. to the right and 2 cm. to the left of the median line, and downward 5 cm. from the episternal notch. During expiration the edge of a firm body was quite palpable directly above the left sternoclavicular joint. This was supposed to be an enlarged thymus gland producing pressure on

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the trachea, and the question of operation was discussed with Dr. Bell, the surgeon. The condition of the child, however, was deemed to be so grave as not to warrant any operative procedure.

During the night the attacks of dyspnea became still more

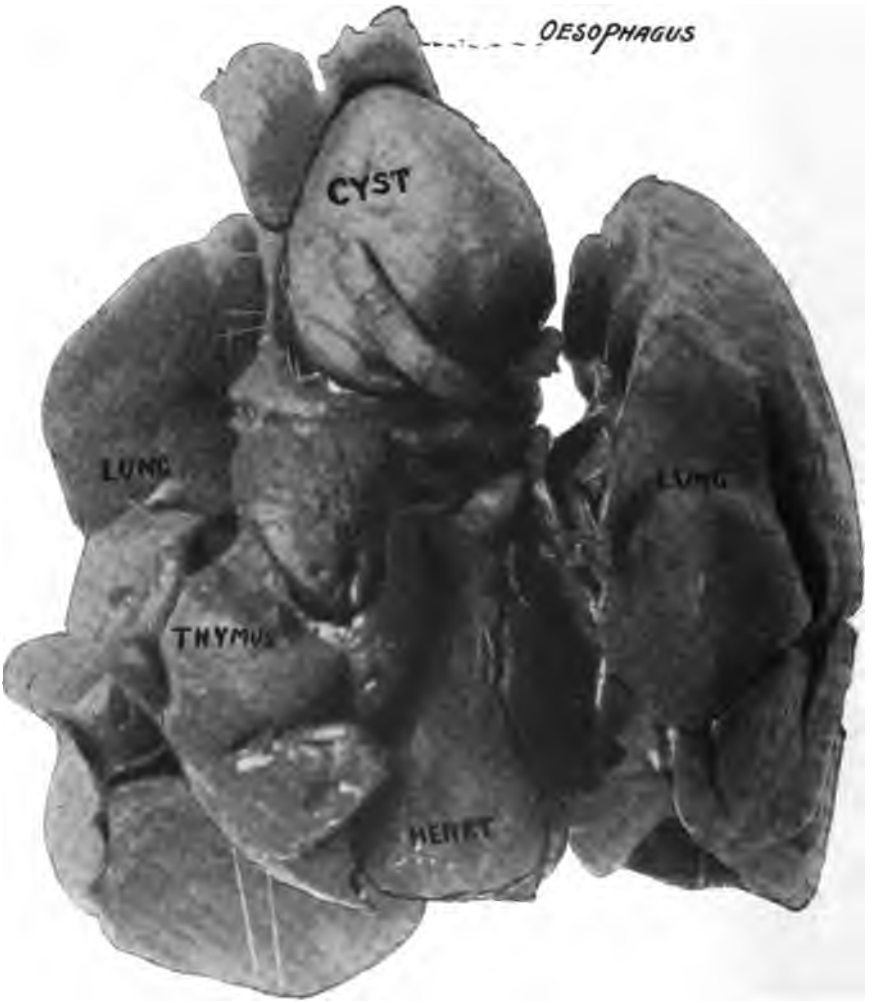

FIG. 1.—Front view. Specimen was mounted on a glass frame in its exact anatomical position, so that the photographic view is same as looking into the opened chest.

frequent. A small dose of morphia given hypodermically appeared to give relief, and the child fell asleep for two hours. At four o'clock, during a more than usually severe attack, the respiration ceased entirely, but, under stimulation, again started.

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Cardiac stimulants were now administered, but death took place three hours later.

Permission was obtained from the parents for a limited autopsy. This was performed by Dr. John McCrea, of the Royal

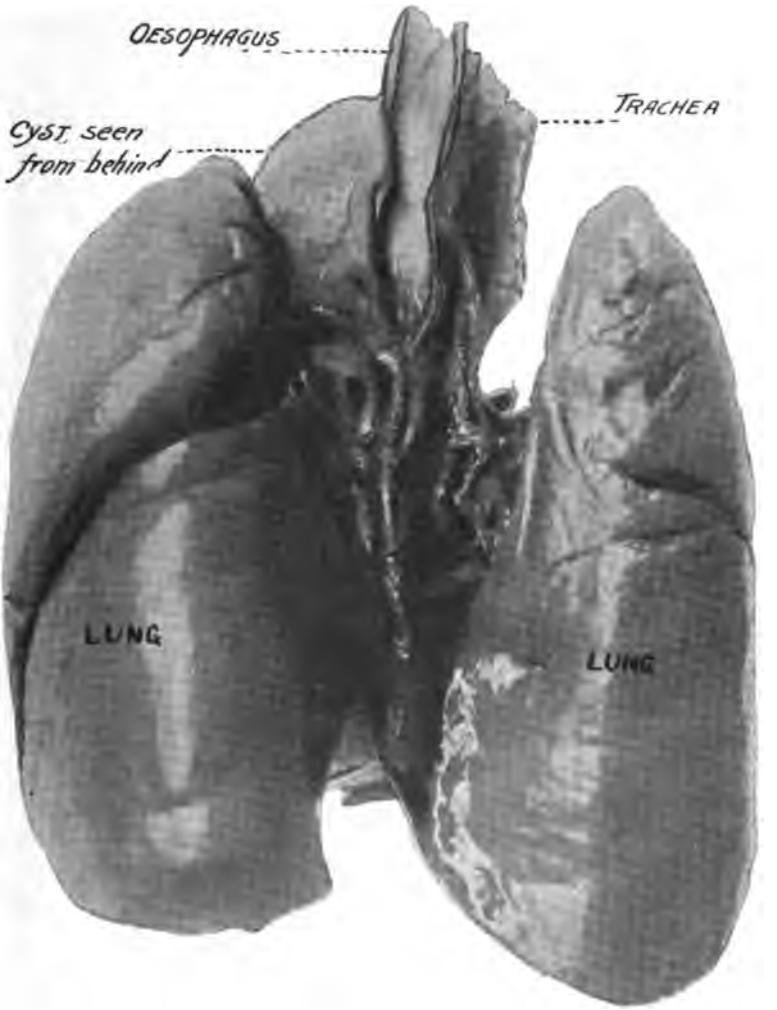

FIG. 2.—Back view, showing the relation of the esophagus and trachea to the posterior aspect of the cyst.

Victoria Hospital, the same evening. The following is his report:

"On opening the thorax numerous adhesions were noted between the top of the sternum and the thymus gland. The thymus gland itself was very prominent; it measured 5 cm. long by 3 cm. wide, and was bifurcated below. It reached as low as the fourth

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interspace, namely, half way down the front of the right ventricle. On lifting it up a rounded, yellowish cyst was seen to lie behind it. The cyst measured 4.75 cm. x 4.5 cm. x 3.5 cm.; was roughly globular in shape and in places a little bossed, as if multilocular. Its upper edge reached about 1 cm. above the sternal notch, or slightly higher, scarcely reaching the lower edge of the thyroid gland. It lay partly behind, but mostly to the left, of the trachea, and partly in front, but mostly to the left, of the esophagus, so that its right side projected between these two, pushing one forward and the other back. Looked at from the front it reached 1½ cm. to the right of the trachea and 3 cm. to the left. It lay behind the thymus and appeared to project downward against the left auricle. Coursing over the front of the cyst were the innominate and the carotid arteries, and the left pneumogastric nerve. These were all held loosely by connective tissue lying on the sac wall.

"No pedicle could be found, nor could any connection be seen that was not superficial to the surrounding organs. Both the trachea and the esophagus were normal on opening. The lungs were bulky and emphysematous. Both pleuræ and pericardium were normal.

"On opening the cyst it was found to contain about one ounce of clear, viscid mucus-like fluid, of the consistence of the white of egg. It was unilocular, with imperfect subdividing walls inside. On section, the wall was found to be muscular and fibrous; the muscles lay in two layers, at right angles to each other; the inner was circular and the outer was longitudinal. Inside of this was connective tissue, while the inner surface was lined by well-defined columnar ciliated epithelium."

After a careful research throughout all the available literature at our command, we can find no report of any case quite similar to this. At first it was deemed to be a branchial cyst. Branchial cysts, however, are situated, as a rule, laterally in the neck, 2 to 3 cm. above the level of the clavicle, and near the inner border of the sternomastoid muscle. Kaufmann states, however, that a cyst representing the fifth branchial cleft may have a position in the median line low down, just above the sternal notch; nevertheless, its connections would point upward toward the root of the tongue. Such a cyst would be lined with mucosa covered with ciliated epithelium.

Ziegler, in his text-book of special pathology, states that cysts

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may arise as a consequence of an imperfect closure during fetal life of the communication between the respiratory passages and

FIG. 3.—Congenital absence of esophagus in upper half. Pharynx above ends in a blind pouch. Lower half of esophagus communicates with trachea at bifurcation. (From a specimen in the Pathological Museum of McGill University.)

the esophagus. Such communication usually has its site above the bifurcation of the trachea, and may occur when the two ends of this communication close and the passage itself remains as a cyst.

Two other methods of origin have been described: One in which rests are pinched off from the trachea or from one of the bronchi; possibly in some cases representing a portion of an accessory or rudimentary bronchus. In other cases they appear to arise from dilatations of mucous glands in the wall of the trachea. Such are said generally to occur on the posterior wall of the trachea, and may be as large as a walnut and protrude into the space between the trachea and esophagus.

The relation our cyst bore to the surrounding tissues, together with the absence of cartilage, and the character of its walls with two distinct layers of muscular tissue, all point to the probability of its origin in a partial persistence of the original fistulous communication between the trachea and esophagus; its character

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also differentiates it from any dilatation of a mucous gland, or rest pinched off from the trachea or bronchus. Although both Kaufmann and Ziegler refer to the occurrence of mediastinal cysts of the origin described, we are unable to find any similar case to our own reported.

Cysts of the mediastinum are reported by several writers, but have little resemblance in their details to this one.

Stilling reports a cystic tumor in the upper part of the anterior mediastinum in a man of forty-five years, who died of cerebral hemorrhage. The cyst was the size of a hen's egg, and lay under the arch of the aorta, only lightly bound down. In its walls were mucous glands, cartilage and basal membrane resembling that of trachea, and it was lined with ciliated epithelium. It thus appears to have arisen from a pinched off rest from trachea or bronchus.

Virchow relates the case of a mediastinal cystic tumor in a man of twenty-two years, which produced no cough, dyspnea or pain. It was in part sarcomatous and in part cystic. In the walls of the cyst were muscle bands, mucous glands and plates of cartilage lined with ciliated epithelium. He regarded it as a teratoma myomatodes.

C. Henning gives the details of a cystic growth found in a stillborn babe. The cyst was of the size of a plum, near the esophagus, extending between the level of third and fifth cervical vertebræ. Its origin is not discussed.

Kurz reports a case of diverticulum of the esophagus, which, when full of food, compressed the esophagus and rendered swallowing difficult. Washing out this diverticulum at once gave relief and the child improved.

Riegel states that in the 42 mediastinal tumors spoken of in literature, 5 were so-called "dermoids"; but all his cases were of adult age.

The literature of mediastinal cysts, however, is extremely scanty, and we have found none in the literature at our disposal which bears any close resemblance to the one we have described.

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POST-TYPHOID DELIRIUM.

BY ALLEN BAINES, M.D., C.M., ETC.,

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The status known as post-typhoid delirium is very seldom met with in children. In looking over the various hand-books on Pediatrics in my possession, I find the condition not noted at all, or very casually referred to as being uncommon.

Dr. Samuel Adams, of Washington, reported 4 cases at the meeting of the Pediatric Society in May, 1906, giving a clear and concise clinical report of each case. Dr. Adams termed it "Post-typhoid Mania." In speaking of my cases to Dr. Ernest Jones, Associate in Psychology at the Provincial Hospital for the Insane, Toronto, he demurred at the term "mania" and said, regarding my cases: "In the case of any mental disturbance arising during, or in relation to, an acute fever, such as typhoid, the immediate problem that arises is to decide whether it belongs to one of the two following fundamental different conditions:

"(*a*) Psychoses, in which the essential cause is the poisoning of the cortex. This condition would be called a toxic, or, more specifically, a typhoid psychosis. It may arise either during the fever (*e.g.*, many cases of delirium) or after (then often wrongly called post-typhoid neurasthenia).

"(*b*) Psychoses due essentially to deeper disturbances in the personality, where the fever has acted merely as an exciting cause of the outbreak, such as an accident; so may operate.

"Here the psychoses may be of any form, according to the more fundamental causes, and the diagnosis as to which form, or as to whether the condition belongs to group (*a*) or group (*b*) depends upon an exact study of the mental symptoms. Without knowing more about your cases, one could not decide as to the diagnosis, but from what you tell me I should strongly suspect that they belong to what we call 'anxiety hysteria,' the most frequent psychoneurosis in children. I have seen a number of such cases in relation to typhoid. Lastly I have to say that it would be quite improper to use the term 'mania' in connection with any such

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conditions. 'Mania' is a specific disease, or rather a form of a specific disease (maniacal depressive insanity), which rarely occurs in children at all (the youngest case I have seen being fifteen years of age). Further the term 'maniacal' is even less appropriate. It is not necessary to remind you that the term is very far from being a synonym for screaming or excited."

This reasoning on Dr. Jones' part seems so clear and lucid that, coming from such a man, it lends weight to my altering the term "mania" to "delirium."

An article by Dr. J. Lovett Morse, published in the *Boston Medical and Surgical Journal,* February 27, 1896, in which 282 cases of typhoid fever in children are carefully analyzed, is probably the most exhaustive and instructive compilation on this subject to be found. Every symptom, complication and sequela is carefully considered in his own cases, and those of a vast number of well-known observers all over the world. The digest made of this mass of clinical information and the conclusions therefrom drawn are of a most valuable and reliable character. Too great time would be taken up in quoting much from this monograph, but a few excerpts as to acute delirium may be not out of place. No mention is made as to the subject matter in hand, viz., post-typhoid delirium, but as to delirium, he quotes:

Earle says: "In severe cases symptoms are very pronounced and meningitis simulated. Parrot thinks that stupor is rare and coma almost never occurs and that acute delirium is more common, and explains this by the undeveloped mentality of children. Henoch, Strumpell, Earle and Wilson all agree that nocturnal delirium is often replaced by continuous and causeless screaming. These opinions are, however, applicable only to the delirium during the fever."

CASE I. Frank S., aged six years, born in China, the son of a missionary, parents English, admitted to Hospital for Sick Children, Toronto, October 25, 1909. Previous history, pertussis at one year of age; scarlet fever one and one-half years; dysentery twice, at eight months and at one year; lumbricl from five years and some seen recently.

Present History.—Eight days ago had a chill and sharp attack of vomiting, followed by pain in back of neck and general listlessness. Bowels constipated, spleen enlarged, a few pale rose spots on abdomen and flanks, mentally clear. Appeared to be a particularly bright, cheerful child.

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October 17th, began to show signs of drowsiness and marked lethargy; temperature had been high all the time, ranging from 104° to 106° F.

October 21st, condition about the same, pulse softer and poor tension. Tympanites more marked.

November 4th, the condition remained about the same; temperature lower, running 99° to 102° F.

November 8th, temperature normal. He is quiet and does not speak, takes nourishment well.

November 11th, doing very well until midnight, when he began yelling and kept on doing so until he could yell no more, his voice having disappeared. This state of affairs went on with varying intensity, the child often being quiet for an hour or two, but when spoken to or given nourishment, starting off with a vigor simply marvelous and extremely disturbing to others in his immediate neighborhood. It was not until November 19th that any marked improvement took place. The night nurse reported a quiet night and that he answered "yes" or "no" three or four times to questions. It was not until December 10th that he was really quite normal. It will be noticed that his temperature became normal on November 8th. On the 11th delirium of a maniacal character set in and lasted unintermittingly until December 10th, a period of over four weeks. From this time on the case presented no points of interest. It was one of steady convalescence, and he left the hospital December 22d, very fairly well. No drug or treatment of any kind seemed to have a quieting effect, or if it did, it was of such an evanescent character as to be hardly worthy of notice.

It is unnecessary to give a full history of the case, it being manifestly an ordinary attack of typhoid fever, having all the classical symptoms and verified by positive Widal and diazo reaction. I therefore give a few notes at some days' interval.

CASE II. Duncan M., age six years, admitted to hospital August 23, 1909, family and personal history unimportant. The boy was ill for two weeks before admission, having had frequent attacks of vomiting, loss of appetite, feverishness and general malaise. Physical examination showed nothing noteworthy. Temperature 102° F., which slowly receded to normal on September 8th, sixteen days after admission, probably thirtieth day of fever. On September 8th had a somewhat severe convulsion lasting fifteen minutes. There was a slight rise of temperature at this time, receding to normal next day. On September 21st he started

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screaming at the top of his voice. No remonstrance had any effect. Small doses of codein had to be given at night to quiet him and for the sake of other children in the ward. Took nourishment very well. On the 23d, the phase of delirium changed somewhat; any remark made to him he would repeat over and over again, such as "Are you better Duncan?" as loudly as he could yell; at the same time his face became distorted as if in acute pain. On the 30th he became much quieter, having yelling attacks only three or four times a day, but was still very irritable. By October 6th, forty-four days after admission, he was quite normal, intelligent, quiet and bright. From this date convalescence was uninterrupted.

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THE CASUISTRY OF CHYLOUS ASCITES AND CHYLOCELE IN SUCKLINGS AND VERY YOUNG INFANTS, WITH REPORT OF A CASE.

BY DAVID MURRAY COWIE, M.D.,

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Reports of cases of chylous effusions into the serous cavities have greatly increased since Dr. Samuel Busey's first collection of 53 cases in 1889. Dr. Busey's report covers the period from 1699 to 1889. Since this time further collections have been made, notably by Bargebuhr in 1893 and in 1895, by Verdelli in 1894, by Rotmann in 1897, and by Shaw in 1909. A few other collections have been made, but either because of incompleteness or unsatisfactory arrangement with regard to references they are not referred to. Since Shaw's collection quite a number of new cases of chylous ascites have been recorded. These may be easily found in the Index Medicus or Bibliographia Medica.

A case of chylous ascites and chylocele in an infant fourteen days old, entering my service at the University Hospital, prompted me to look up the occurrence of this condition in sucklings. To this end the above-mentioned collections were gone over carefully, as well as other references, during and since the period covered by these reports. As I have found several cases of chylous effusions in adults during the period covered by the above reporters and evidently overlooked, it is not at all improbable that I, too, have failed to find recorded cases in infants.

In the collected cases frequently no age is given. I have endeavored to ascertain from the original articles or abstracts of them whether these cases were in adults or infants. One or two cases recorded as children in these collections were found to be in adults. In all I have been able to collect 8 cases in infants, which, with my own, makes a total of 9 cases recorded since 1691, when Dr. Morton reported the first case. I have arranged these in two groups. Group I. includes 6 cases during the suckling period. All but one of these are under twelve months old. Group II. includes 3 cases in infants up to the end of infancy, as pediatricians regard that term, the oldest being thirty-two months.

The reports of the 9 cases are given in full. For the sake of brevity I have made some rearrangements and have omitted

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many unnecessary repetitions. The reader is particularly directed to the first and very interesting case of Dr. Morton, in the second group, published in 1691. For the excellent translation of this from the Latin I am indebted to Professor Meader, of the Latin department.

Chylocele, chylous effusion into the scrotal sac, was present in 4 of the cases—those of Kaminski, Letulle, Kerr and Cowie. In the latter 2, fluid was withdrawn by paracentesis and could be displaced into the abdomen by pressure. Letulle simply makes the statement that the scrotum was tumefied.

GROUP I. CHYLOUS AND CHYLIFORM ASCITES IN SUCKLINGS.

CASE I. Wilhelms, 1874, in infant ten months old.

Mail-carrier Gierling and his wife, both well and robust, were rejoiced at the birth of a strong girl baby on New Year's night, 1873. When the child was six weeks old it developed whooping-cough and, in spite of all that could be done, the child remained ill for two months. About two months after recovery from the whooping-cough the parents observed that the abdomen of the child was gradually increasing in size. The child took its food well. Finally the abdomen became so distended that it vomited. The doctor in charge of the case gave up all hope of recovery and withdrew. [The case came into Wilhelms' care first of October, 1873.] There were all the signs of ascites in this six-months-old baby. Because of this I was likewise moved to discourage the parents in any hope of its recovery. Operation was necessary. I made a paracentesis abdominis and emptied, to my great astonishment, a white fluid, 16 pints in all (16 Schoppen maas). The fluid had the appearance of milk. Prof. Rindfleish, of Bonn, declared, after examining some of the fluid, that it was pure "lymph" that had flowed into the abdominal cavity from the thoracic duct. Examination of the abdomen after tapping.—The organs were in normal condition. In the region of the navel a tumor mass the size of a large hen's egg was felt lying attached, immovably, to the vertebral column. My opinion is that the tumor is nothing more than a lymph gland, which is connected by a fistula with the thoracic duct, in consequence of which the lymph is permitted to flow into the abdominal cavity. [During a period of seven months, from October to April, Wilhelms tapped the abdomen ten times.] Shortly before the operation, when the ascites was at its height, and the day following the

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tapping, the child always suffered from vomiting and took almost no nourishment. [No further record of case.]

CASE 2. Winiwater, 1877, four months old.

Johanna L., daughter of a day laborer living in poor circumstances, was born April 20, 1876, the seventh child of a weakly woman grown old before her time. The brothers and sisters of the infant all died either at birth or in early infancy. Reporter says nothing about possibility of lues.

Immediately after the birth of this infant the mother and midwife noticed that the baby's abdomen was unusually large. The physician in charge made the diagnosis of abdominal tumor and said the child could not live. It continued to live, however, took the breast greedily, and developed slowly. The abdomen gradually increased in size, so that at four months it was so large as to interfere with respiration and retention of food in the stomach. There was a tendency to constipation. Vomiting occurred frequently, particularly if she took her food too rapidly.

August 22, 1876, at the age of four months, the child entered the out-patient department of the Kronprinz Rudolph Kinderspital, in Wein.

Status Presens.—Baby is thin, musculature is poorly developed, face is cyanotic. The thorax circumference is greatly increased. The abdomen is large, 65 c.m., tightly stretched, not symmetrical. The right hypochondrium is distinctly fuller than the left, but no sharp boundaries can be made out. Because of the distention palpation is impossible. The percussion note is tympanitic all over the side opposite to that upon which the child lies. There is dullness in the flanks, the exact boundaries not made out. There was no edema of lower extremities. The urine showed no albumin.

Immediate paracentesis was necessary. A trocar was introduced at a point near the left hypochondrium, where there was complete dullness. After the removal of the stylette a white fluid poured from the opening, to the great astonishment of all present. The fluid resembled milk in appearance and was the most perfect substitute one could think of—the same color, the same consistency, yes, even the same smell as fresh milk. Three liters were obtained and the trocar removed before the abdomen was completely empty. Although the fluid was thus reduced the inflation of the intestines was so great as to make examination of the abdomen difficult. The greatest prominence was still in the

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right hypochondrium and the dullness reached downward from the liver obliquely across the middle portion of the right lobe of the liver through the remaining fluid. There was no change in appearance when she lay on her side. At this point, on deep palpation, a movable mass could be felt, which seemed to be composed of several parts (folds) resting on the vertebra, of the consistency of a flabby cyst. Fluctuation indefinite. Impossible to determine if tumor is attached to the liver. Upper border of the liver palpable. The mass is free from the abdominal wall and does not move on respiration.

Tappings were made as follows:—

August 22, 1876	3 liters
September 12, 1876	2½ "
November 18, 1876	Amount not given
January 16, 1877	" " "

Before each tapping the child's condition was always bad; always improved after tapping. At the last tappings a "stronger" trocar was pushed through the left epigastrium and the abdomen emptied almost completely. One could now recognize the tumor plainly. Its composition was that of a soft, yielding sac. [No further account of the progress of the case is given.]

Examination of the ascitic fluid was made by Professor Ludwig, and is as follows: The fluid has no odor. It has the appearance of milk. The reaction is alkaline. On standing a long time fibrin flakes settle to the bottom of the vessel. A layer of cream lies upon the surface. The specific gravity is 1,012. The chemical analysis:—

Albumin	45.00	parts
Fat	36.80	"
Sugar	0.20	"
Chlorides	3.41	"
Sulphuric anhydride (SO_3)	0.23	"
Phosphoric anhydride (P_2O_5)	0.14	"
Phosphoric (acid) alk. earths	0.01	"
Carbonic anhydride (CO_2)	0.484	"
Carbonic (acid) alk. earths	0.099	"
Potassium	0.24	"
Sodium	2.85	"
Calcium	0.077	"
Magnesium	0.016	"

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CASE 3. Nieuwondt and Rozenzweig, 1892, in infant fifteen months old.

A case of milky ascitic fluid in a female infant fifteen months old, living in Cape Town, South Africa. May 24, 1889, he first saw the case. The child was very thin and weak, and was suffering from an attack of diarrhea and vomiting. May 30th she had recovered. June 10, 1889, she became restless and seemed to be in pain. June 11th the abdomen became distended and was very tender; tympanites. The pulse was rapid and wiry. The temperature was normal. June 20th the symptoms had become less acute. Dullness could be detected in the lower portion of the abdomen. June 28th the symptoms were practically the same, but the abdomen continued to increase in size and fluid rose in the abdomen, causing it to bulge beyond the ribs. There was now cyanosis, fever of 102°F., sweating and diarrhœa.

The case was now thought to be one of tuberculosis, tabes mesentericus. On September 25, 1889, the abdomen was tapped and 20 ounces of white, milky fluid aspirated. In two months' time the infant had been aspirated eleven times, and in all 680 ounces of the milky fluid had been removed. After this, November 5th, the symptoms abated and the child continued to gain in weight until January, when it was strong and healthy. In six months more she was above the average for her age. The author is inclined to believe that there is evidence of three distinct attacks of peritonitis in this case and that the milky fluid was due to this cause and was not pure chyle. [The child evidently got well.]

Summary of tappings, etc.:—

September	5,	1889,	20	ounces.	Did not completely drain.
"	9,	"	40	"	
"	16,	"	80	"	Completely drained.
"	24,	"	90	"	Abdomen larger than ever. Child thin, fretful, anxious-looking, sweating freely, appetite good when not distended with flatus. Three to four loose bowel movements daily.
"	29,	"	55	"	Did not drain completely.
October	4,	"	90	"	
"	10,	"	50	"	
"	14,	"	70	"	
"	20,	"	70	"	October 26th, diarrhea.
"	28,	"	60	"	
November	5,	"	55	"	November 9th, diarrhea.
"	14,	"	..	..	Little change in abdomen, symptoms improving. See above.
			680	ounces,	20,400 c.c.

Analysis of Fluid.—The fluid appeared like rich cream of milk. Specific gravity, 1,022. There was no spontaneous coagula-

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tion. Neutral. Faintly sweetish odor. No pinkish discoloration. Heavy coagulation on heating. Cold nitric coagulates and when heated turns yellow. KOH increased the fluidity. Microscopic examination showed a finely granular matrix, with large nucleated cells "bearing on the whole but slight resemblance to the general character of chyle." No fat estimation made. Large amount of fat present.

CASE 4. Kaminski, 1895, in infant five weeks old.

Stanislaus G., five weeks old. Normal birth. Both parents well. Father robust, mother a well-built woman. Father denies lues. No tuberculosis on either side. Hygienic conditions in which parents live are good. Parents could give no cause for abdominal swelling. There had been no injury. This infant was the first living child to these parents. He had been well cared for, but, as they say, the children in their family do not thrive. The cause of death in their children they were unable to state. After the birth of this child profuse suppuration of the navel occurred. It is now healed.

The child is breast-fed and has a good appetite; three to four stools daily, large and good, sometimes green. A few days after birth the mother and midwife observed a large bloating in the child's abdomen, which continued to increase until it was seen by Kaminski at five weeks old.

Status Presens.—Normal build. Well nourished. No fever. Drinks well. Lung borders normal. Breathing vesicular all over. Slight dullness in the bases of the lungs behind. Heart borders normal, sounds clear and fairly strong. Pulse, 140. Respiration, 48. No edema of extremities or abdomen. The navel is healed, protrudes so that it forms a hernia. The abdomen is moderately distended in all dimensions, as is also the epigastrium. Surface is smooth and glazed, small varicose veins showed through the skin. Liver and spleen not felt. Percussion gave a tympanitic sound over upper portion of abdomen as far down as two fingers above navel; below this, dullness. Fluctuation determined with child on its back. Contour of abdomen and the dullness changed on position. Sitting or standing the abdomen protruded, etc. There is a collection of fluid in the scrotal sacs; they were almost twice the normal size. No fluctuation. No pain.

The general condition of the child seemed good, but the distention became so large that tapping was necessary. This was

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done between the median line and the anterior superior spine. "To our astonishment we got, instead of a purulent fluid, a whitish-yellow fluid, which had the appearance of milk, chylous or adipose ascites. 750 c.c. were obtained. The abdomen diminished in size, the fluid in the scrotum disappeared, it drew together and the veins in the abdomen became much smaller." The fluid soon reformed in the scrotum and the abdomen gradually filled up.

Careful clinical notes of the temperature, pulse, respiration, and of the changes in size, and dull areas of abdomen were made until the middle of August, the last time it was seen by Kaminski. At times the distention became so great as to cause cyanosis and vomiting. At other times the fluid seemed to have decreased in amount. Now it seemed to be perfectly well and there were no signs of fluid in the abdomen. Urine examination several times always negative.

Examination of the Fluid.—The fluid is white, translucent and looks like milk. Specific gravity, 1,007. Reaction slightly alkaline. Coagulates three days after tapping. On the fourth day large curds form at the bottom of the vessel the size of beans, room temperature. No decomposition in four days. Fifth day an odor began. At no time did cream appear on the surface, ten days. Under the microscope many small, highly refractive bodies and some larger fat globules resembling those found in milk are seen. Very seldom does one see such small lymph cells. Ether causes the fat globules to flow together and form larger and smaller fat cells. The clots proved to be network of fibrin threads, in the meshes of which the granules and globules had been caught, as well as a few red and white blood corpuscles. No tubercle bacilli were found.

Chemical analysis:—

Albumin	2.297	per cent.
Fat	4.756	" "
Sugar	0.000	" "
Chlorides	0.195	" "

Discussion.—Kaminski was at first inclined to think the case due to lues, because ascites in young infants, fetal ascites, so frequently follows in its wake. The second supposition was that the child in consequence of its diseased navel had developed peritonitis. The nature of the fluid led him to abandon these ideas. He gives no comment as to the probable or possible cause of the

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chylous fluid. In the clinical notes no records of abdominal palpation are to be found, only percussion and measurements.

Treatment consisted in the application of a plaster composed of equal parts of Ung. cinereum and emplastrum hydrargyri to the abdomen. For the weak heart action, at times he gave wine. Because of digestive disturbance, vomiting and diarrhea, hydrochloric acid.

CASE 5. Kerr, 1909, in infant sixteen days old.

Kerr reports a case in the *New York State Journal of Medicine,* January, 1909, in a child sixteen days old, as follows: Male, sixteen days old. Polish parentage. Family history negative. Father and mother in perfect health. Full term baby. Normal labor. Child in all respects normal at birth. Third day of life, after breast-feeding had been instituted, mother noticed an enlargement of infant's abdomen, which progressively increased until the sixteenth day of life, when it was admitted to the hospital, August 24, 1908.

Physical Examination.—Infant anemic, musculature flabby. Head normal size and shape. Fontanels widely open, no tension. Oral cavity normal. Well-marked fissures at angles of mouth. Slight discharge from nostrils, excoriating upper lip. No edema. Circumference of chest 13 inches. Examination of chest negative. The abdomen is considerably enlarged, circumference 19¾ inches. Wall thin, skin smooth, veins not prominent, navel healed. Palpation impracticable. Percussion.—Lateral areas of dullness with tympanitic area in the center of abdomen. Dull areas change with position. Scrotum is swollen evenly, the size of a baseball. Penis buried in swelling and cannot be brought into view. The scrotum is soft, becomes tense while infant cries. No impulse. Position has no bearing. Reduction in size on continued gentle pressure. There are two small anal fissures.

Directly after examination scrotum was tapped and 15 ounces of rich, milky fluid were withdrawn. The abdomen became lax. Fluid continued to flow from the puncture for several hours, estimated at 3 ounces. Upon admission temperature was 98.2°F., pulse, 98; respirations, 24. Twelve hours after tapping temperature was 100°F. Pulse and respiration remained the same.

Clinical Examination of Fluid.—White, alkaline, odorless.

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Specific gravity, 1,010. Profusion of fat cells and a few leukocytes. Urine examination negative.

Following the tapping the infant progressed well until the 27th of August, when the nasal discharge became profuse, lasting twenty-four hours. The infant slept soundly and cried but little. Nourishment, whey.

A rash appeared on the abdomen August 29th, having the characteristics of syphilitic eruption.

Five days after the first tapping another scrotal paracentesis was done, and 8 ounces of similar fluid were withdrawn. The infant continued to progress well. It was decided September 5th to drain the remainder. Twelve ounces were obtained.

Examination of Fluid.—Milky white, odorless, specific gravity 1,013, alkalin. Shaken with ether and filtered, filtrate is slightly opaque. Microscopic examination.—A profusion of fat cells, a few cholesterine crystals, and a few blood corpuscles, undoubtedly due to wound. Fluid has characteristics of chyle.

Urine Analysis.—Colorless, like water clouded with milk, no odor, cloudy white sediment, faintly acid, specific gravity 1,003. Urea 0.001, sugar negative, albumin trace. Indican negative. Microscopic examination.—Numerous cholesterin crystals, profusion of fat cells and very few bacteria.

September 6th, a double inguinal hernia appeared, which reduced by posture. September 19th, infant improved in general appearance, abdomen soft. September 30th, infant still improving. Scrotal sac greatly reduced in size. Small gain in weight. Stools normal. October 31, 1908, infant still alive and improving.

CASE 6. Author's case in infant seven weeks old.

Russell B., from Durand, Mich., entered the Pediatric Clinic of the University of Michigan Hospital May 27, 1909.

History.—The first born to these parents. Mother, twenty-two years old, has always been healthy, but looks pale, anemic at present. She began to menstruate at thirteen. Always regular, no miscarriages. No specific history obtainable on either side. The father is a strong, robust man. Family history negative. About the eighth week of her pregnancy the mother was confined to her bed for two weeks on account of vomiting and headache, no chills or fever, no hemorrhage. The balance of the pregnancy was uneventful until March 15, 1909, when she was seized with

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pains which were interpreted as beginning labor. Her physician was in attendance most of the night. From this time until April 8th, when the child was born, she continued to have pains which simulated labor pains. These became more marked on April 8th. Her attending physician found the cervix undilated. He instituted manual dilatation, ruptured the membranes and delivered with forceps. Presentation was normal. Child had to be resuscitated. The birth weight is said to be 12 pounds, and the abdomen to have been markedly enlarged. Two weeks after birth the mother noticed the abdomen was growing larger.* No special attention was given to this until the fifth week, when the child began to vomit. Since which time the abdomen has not appreciably increased in size. With the exception of the gradual enlargement of the abdomen the child has seemed perfectly well since birth. He nurses well, has from one to five stools daily, which the mother says always contain curds. His weight has increased markedly, so that now, at seven weeks, he weighs 16 pounds. He has no pain, does not cry, seems happy, belches considerable gas, but passes little flatus. He has what the mother calls blue attacks, blueness of the face and neck and at times of the upper and lower extremities, particularly if he is exposed to cold.

FIG. 1. *Author's case.* Seven weeks old. Chylous ascites and chylocele.

Patient was first seen by Dr. James A. Rowley, of Durand, May 26, 1909, who sent the child to me for examination and diagnosis. The parents would not permit the infant to enter the hospital, and returned home on the first available train after my

* Doubtless chylous fluid was present in the abdomen at this time.

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preliminary examination. I succeeded, however, in obtaining considerable data concerning the case. I am indebted to Dr. Rowley for obtaining many points in the history which I was unable to secure during the child's short stay in the clinic, and also for his watchful care and treatment of the case to the present time.

FIG. 2. *Author's case.* Seven weeks old. Showing stomach pushed upward and forward.

Status Presens.—May 27, 1909. Patient is 23 inches long, circumference of head 15¾ inches, around the nipple 16½ inches, epigastrium 21 inches, around navel 22 inches. Well-nourished child of average sized skeleton, well-developed musculature, panniculus moderately thick. There is a dusky appearance of the face, neck and ears (cyanosis, but not marked). The mucous membranes are clean and of fair color. There are no enlarged glands, no edema; there is a fatty bunch an inch in diameter on the dorsal surface of the left foot, at base of toes, of the consistency of normal muscle tissue. Circumference of foot at this point 4¾ inches. The right foot measures 4 inches at similar point. Temperature normal, pulse normal. Respirations are slightly accelerated and shortened.

Further examination was carried on under anesthesia. The chest is of average size, the lower border of ribs flaring as if pushed up by intra-abdominal pressure, symmetrical; expansion is slight, symmetrical. Percussion is negative. Auscultation negative. No increase in the heart boundaries, no murmurs. Heart shock not felt, rhythm regular.

Before anesthesia the abdomen was symmetrically enlarged and bulged in the flanks. Just below the ensiform in the median line is a protrusion 1½ inches in diameter, soft, tympanitic. When the patient cries it becomes tense. There seems to be some

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separation of the recti at this point. The entire abdomen is tense, unyielding to pressure. On palpation it gives the sensation of large masses, which are firm, rubbery and smooth to the touch. There is a marked flatness in the flank, with dull tympany above the navel, down to 1 finger below the navel; fluctuation very questionable; no change in dullness with position of patient.

FIG. 3. Babcock test, showing per cent. of fat (7.2) in chylous fluid. *Author's case.*

Under anesthesia the walls became slightly relaxed. No tumor masses could be felt. Kidneys, liver and spleen not felt.

The scrotum is enormously enlarged. Left side larger than right. Greatest circumference 9½ inches. The whole mass has a water bag feel, is tense, but on gentle and continuous pressure can be almost completely reduced, to return again when the pressure is released. Testicles could not be felt. Later Dr. Rowley reported them descended.

Radiograph of body, with colon injected with bismuth water, shows normal colon. No evidence of tumor masses in abdomen or thorax. One plate suggests fluid.

Abdomen was tapped midway between navel and right superior iliac spine; 15 c.c. of rich milky fluid were withdrawn, having no odor. After the radiograph was made the colon was flushed with water to remove as much bismuth as possible. When the milky fluid came into the syringe the nurse, fearing it was bismuth solution, reproached herself, assuring me that she had washed all the bismuth out. However, the fluid, as I had suggested, proved to be chylous in nature, an analysis of which is given below.

Sample removed May 27, 1909: 15 c.c. rich milky fluid, no odor. Albumin, heavy precipitate with heat and nitric acid test ½ by volume. Sugar, positive test with Fehling's solution. Treating with sulphuric acid fat globules rise to the top.

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The fluid becomes opalescent on treating with KOH and extracting with ether. Microscopic examination shows emulsion in fine subdivision, granular in appearance, the granules in active Brownian motion resembling micrococci. Fat, 9.2 per cent.

Sample removed by Dr. Rowley May 31st, received June 1, 1909. Rich milky material of the consistency of rich Jersey whole milk, slightly alkaline in reaction; specific gravity, 1,004; sugar, positive to Fehling and Nylander reagents after standing for fifteen or twenty minutes. Treating with KOH and ether it soon becomes opalescent, and after twenty-four hours is perfectly clear. Fat, 7.2 per cent., clear, yellow fat, Babcock test. (Fig. 3.) Proteids estimated as heat and nitric coagulable material after prolonged centrifugalization at high speed, 70 per cent. Fibrin, a trace. The fluid remained perfectly odorless for over two weeks and did not coagulate until after three weeks, when white masses the size of peas rose to the top like curds, completely separating from a perfectly clear liquid below. Hydrogen sulphide strong.

Sample from the scrotal sac gave exactly the same fat content. (Fat, 7.2 per cent.)

Tappings:—

May	27,	1909 ...	15 c.c.	(Cowie)	
June	2,	" ...	120 "	(Rowley)	
July	8,	" ...	960 "	"	
"	20,	" ...	600 "	"	
Aug.	3,	" ...	360 "	"	
"	17,	" ...	240 "	"	
Sept.	17,	" ...	45 "	"	(scrotal sac and abdomen apparently empty.)
			2,340 c.c.		

Dr. Rowley examined patient October 27th. Since the last tapping no symptoms have occurred. Weight, 20 pounds. Child looked well nourished.

Inspection, palpation, percussion, abdomen entirely negative. Right scrotal sac at times seemed slightly enlarged. Child is now taking easily digested food at the table—cereal, baked potato, oatmeal, graham wafers. Gains weight more rapidly on this diet. Normal in every respect.

April 27, 1910. Upon my solicitation the child was brought to the hospital for examination today, with the hope of finding some definite cause for the chylous ascites. The child has been

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unusually strong since birth. Held his head up between three and four months. Sat up at six months, but does not stand or bear any weight on feet at thirteen months.

Present weight, stripped, 24 pounds 10⅛ ounces (11,668 grams). Perfectly well, happy, bright child. (See Fig. 4.)

FIG. 4. *Author's case.* One year nineteen days old. Seven months after last tapping.

Examination under Ether. — Length, 30½ inches. Circumference of head, 18¼ inches. Circumference of chest, 18½ inches. Abdomen at navel, 17¼ inches. Anterior fontanel two cm. in diameter. Scrotum, 7 inches in circumference. Right sac slightly larger than left. Normal in appearance. Both testicles felt. No hernia. Panniculus very thick, particularly over extremities. (See Fig. 4.) Skin is of a good color; no edema. Exposure to cold produces blueness of extremities. Mucous membranes good color. Mouth cavity clean. Six teeth—three upper and three lower incisors. Seventh tooth nearly through gum. Joints normal. No enlarged glands.

Chest is narrow above, ribs slightly flaring below. Marked pigeon breast. Expansion good, symmetrical. Good resonance all over—equal. Auscultation and percussion negative.

Heart.—Apex beat neither seen nor felt. Faint heart shock. Upper border, second rib, parasternal line, right border 4 cm.

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to right of midsternal line, immediate percussion, 2½ cm. mediate percussion. Left border inside nipple line 6 cm. from midsternal line. Heart sounds quick but normal. No adventitious sounds. (See orthodiagram, Fig. 5.)

Abdomen slightly below level ribs, symmetrical, soft, easy to palpate. No tumor mass can be felt. Liver, kidney, spleen not felt. No succussion in stomach several hours after food. Complete relaxation. No signs of abdominal puncture.

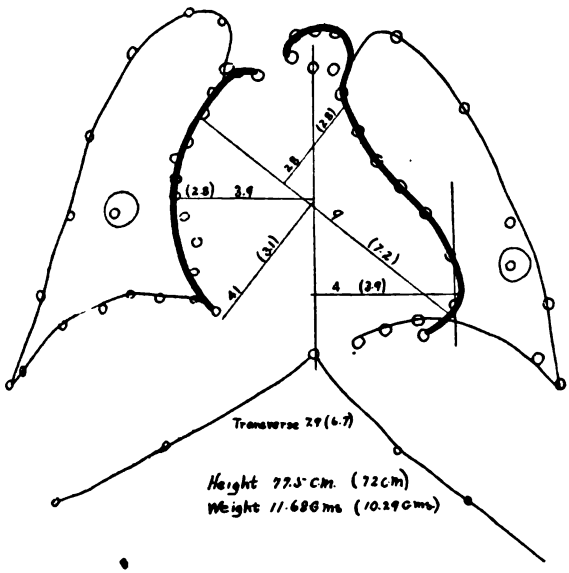

FIG. 5. Orthodiagram of author's case. The figures in brackets are the normal for height and weight indicated, according to Reyher. The other figures are the actual measurements in c.m. in this case. It will be observed that Russel B. is a little taller and heavier than the comparison, which is the nearest I could obtain.

Feeding History.—Breast-fed until tenth month. Then cow's milk diluted one-third with water for two weeks. Then whole milk. At eleven months was given what he wanted from the general table, including bread, cake, pudding, meat, potatoes, apples, bananas, oranges. Craves his bread well buttered and shows marked preference for turnips.*

Discussion.—The history of cyanosis extending not only to

* Final note, April 22, 1911. Child is now over two years old; one year and seven months since last tapping. He is a perfectly well and robust boy. Walks and plays vigorously. His development has been above the average.

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the face, but also to the lower extremities at times, and the increased cardiac area to the right, as shown a year later by the orthodiagram, leads me to believe the chylous ascites was due to pressure of the right heart retarding the flow of chyle into the subclavian vein and the consequent rupture of a chyle vessel below the diaphragm. Cases are on record of chylous effusions due to blocking of the thoracic duct by aneurism (Valsalva and Santorino), to thrombosis of the external jugular occluding the mouth of the duct (Virchow), to plugging of the left subclavian vein by a ragged, long-standing clot (Omerod), and to disease of the right heart producing stenosis of the left venous orifice (Peters). Stewart records a case of dilatation of the lacteals due to fatty and hypertrophied heart with diseased aortic segments and dilated auriculo-ventricular orifices. Roketansky also records a case due to dilated heart from mitral disease.

GROUP II. CHYLOUS AND CHYLIFORM ASCITES IN THE LATTER MONTHS OF INFANCY.

CASE 7. Morton, 1691, in infant two years old.

The son of Thomas Lechmer, about two years old, cutting his teeth, and in consequence of a cold got pneumonia, and for a long time was most miserably treated by an apothecary, who omitted to resort to venesection and other means necessary for a proper cure. Finally, however, through my efforts and those of my excellent colleague, Dr. Croon, although we were called in very late, by means of blood-letting and the application of cataplasms and liniments, as also by the use of lung remedies, he was partially restored. He remained, however, in poor health, with difficult breathing and cough, for the space of at least a year, after which time the poor child began to be afflicted daily with a hectic intermittent fever, and, although this was frequently dissipated by the use of Peruvian bark, yet it always quickly came back, and, as a matter of fact, at varying intervals for a whole year, recrudescences occurring even to the day of his death. From the very beginning of the fever the abdomen began to be distended with a hydropic tumor, which, strange to say, increased daily, with an increase in the cough and dyspnea. In consequence of these symptoms a severe marasmus resulted. Notwithstanding the great emaciation of the body and the acme of the hydrops (which

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should be noted) he retained a robust and handsome appearance of the face, without a trace of sallowness. His appetite was substantial, if anything inclined to voraciousness, up to the very day of his death. From all this I correctly concluded, and to my friends I always expressed the opinion, that the dropsy was chylous, brought about by the draining off of the chyle into the cavities of the abdomen through the lacteal vessels, which had been broken by some accident; and that the wasting which attended it was not really pulmonary, but due to mere inanition, resultant from the withdrawal of the nutritious fluid, due to the body, from the chyliferous ducts, a rupture having been made in these. This was perfectly plain from the outcome.

Tapping the abdomen while the child was still living we extracted several pounds of lactescent chyle, just as sweet (*dulcis*) as is found in the chyle duct itself. From the postmortem examination, when we were not hindered by the difficult breathing and the persistent cough, we found the lungs perfectly healthy, without any abnormal affection, except that in the back part near the trachea appeared very numerous glands, which were quite conspicuous and hard, producing a marked pressure on the chyle duct itself, almost at the point where it reaches the subclavian vein. They were so large and heavy that, on account of the pressure so induced, shutting off the lumen of the duct, it seemed impossible for the chyle to pass into the blood, and thus, beyond doubt, it was brought about that the frail lacteal membranes lying in the abdomen being stretched beyond their resistance (the chyle continually passing and not finding any easy passage above through the chyle duct) at length were ruptured and transmitted into the cavities of the abdomen in a steady stream the chyle which was intended for the nutrition of the blood.

From what I have said it is apparent even to common sense

(1) That the tubercles or pulmonary glands, having been tumefied, arose first in consequence of the bad handling of the peripneumonia on account of the lack of timely remedies and of expectoration.

(2) Just as the annoying dry cough was brought about by the constriction of the air tubes of the lungs, due to the tubercles, so the labored breathing was due to the pressure of the extravasated chyle on the lower surface of the diaphragm.

(3) The atrophy or wasting was not really pulmonary, be-

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cause, although the tubercles were there, still there was no abscess formation and they were not ulcerated, but that the marasmus was the result of chylous hydrops, attended by the steady diverting of this nutritious fluid which should have served for the building up of the body and the nutrition of the blood.

(4) The hydrops was the result of the breaking of the lacteal vessels, situated in the abdomen, and, finally, this breaking of the delicate tissue was brought about by the pressure of the pulmonary tubercles upon the upper part of the chyliferous duct.

CASE 8. Letulle, 1885, in infant thirty-two months old.

A male infant, thirty-two months old, with good family history, lues excluded. No apparent cause for the child's condition. When the child was first seen in consultation, September 16, 1884, it had been ill for about two months with a chronic affection of the abdomen. The child was poor, emaciated, pale, and the subdiaphragmatic portion of the body was very edematous. The scrotum was tumified and the abdomen enormously distended below the false ribs. The umbilicus, protruded by a recent hernia, was easily reducible along with the liquid which filled it. There was marked ascites, as shown by movable dullness. The subcutaneous venous network of the abdomen was distinctly seen and engorged. On palpation and percussion the liver was found to be enormous in size, reaching down beyond the margin of the false ribs and measuring at least 12 cm. on the mammary line. (There was a certain degree of double hydrothorax.) The hepatic gland was hard, very slightly tender to pressure, and pulsated synchronously with the cardiac pulsation. The pulsations were not great in volume and one could not definitely say that there were movements of expansion of the liver. The respiratory embarrassment was great enough to produce orthopnea. The face was pale and slightly cyanosed about the lips and ears. The surface veins of the neck were turgescent, beat irregularly, and could not be counted. The heart beat irregularly, 136 to 140 per minute. No murmur could be heard at the cardiac areas. There was no fever. The appetite was nil. The infant took a little milk and soup with difficulty. The urine was scanty, highly colored, no albumin, no sugar. There were attacks of nocturnal dyspnea. There was no trace of icterus.

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Summary of tappings of abdomen, etc.:—

October	6, 1884,	3.5 liters	yellowish transparent fluid.
November	5, "	3.5 "	thicker, more milky, fluid.
December	3, "	3.5 "	of milky fluid thicker than second. [Fluid evidently had considerable yellow color in it.]
January	28, 1885,	2.5 "	chyliform liquid, clearer and not as thick as two previous samples, having a yellowish white lactescent color, perfectly homogeneous in consistency, with no sediment on standing, and having no odor.

Total amount, 13.0 liters, or 429 ounces.

Chemical and microscopic examination of the last fluid was made and is as follows:—

Specific gravity 13 c.	1,009		
Reaction distinctly alkaline.			
Weight of dry extract at 100 c.	2.010	grams per	100
Weight of fat	0.015	" "	100
Weight of albumin	1.220	" "	100
Weight of salts	0.640	" "	100

The salts for the greater part were made up of chlorides and phosphates. The ether extract after twenty-four hours contained a certain amount of fat.

Microscopic examination showed a few "islands" of leukocytes with fatty granules, which stained black with osmic acid. The fat in the fluid was very finely emulsified.

In discussing the case the author says: "I had under my eyes either a primitive chronic hepatic affection, of which I sought the cause in vain, or an hepatic alteration secondary to a primary cardiopathy probably congenital. The second hypothesis is the more probable one. Given the symptoms presented by this patient, the liver, profoundly troubled in its over-hepatic circulation and affected in its nutrition, was perhaps surrounded by peritoneal adhesions. It is to this explanation that I incline. The physician treating the case found what he thought sufficient proof for the diagnosis of hepatic syphilis. Nothing in the family history or in the present state of the child warranted this belief."

CASE 9. Newcomb, 1889, boy of two years.

This case was never published, excepting in Dr. Busey's report. It is there recorded as a personal communication. No cause could be discovered for the chylous fluid. The fluid contained lymph cells. It was treated by tapping and was still under observation when reported by Dr. Busey. I have been unable to find a final note on the case. No analysis.

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Case No.	Reported by	Date of Report	Age Months	Sex	Cause	Character of Fluid: Total Amount C. G.	Character of Fluid: General Appearance, etc.	Duration Months	Result and Remarks
1	Wilhelms	1874	10	F	Cystic tumor attached to vertebral column. Supposedly a lymph gland connected by fistula with thoracic duct. Lacteal may have been ruptured during a severe paroxysm of whooping cough.	? 10 tappings.	Appearance of milk, "pure chyle."	7	Recovered (?); no final note on case.
2	Winiwater	1877	4	F	Cystic tumor lying on and attached to vertebral column.	5,500 4 tappings.	White fluid resembling milk; no odor; alkaline, creamy layer on top; Sp. G. 1,012; albumin, fat, sugar and fibrin present.	5	Recovery (?); no final report made.
3	Nieuwondt and Rosenweig	1892	15	F	Peritonitis. ?	20,400 5 tappings.	White, creamy fluid with sweetish odor; neutral; 1,022; no coagulation on standing; albumin and large amount of fat present; large nucleated cells in sediment.	9	Recovery; author thinks fluid bears little resemblance to pure chyle.
4	Kaminski	1895	5 W	M	Cause undiscovered; family history might suggest lues, navel infection, peritonitis.	?	Yellowish-white, milky fluid, slightly alkaline, 1,007, coagulated after standing 3 days; albumin, 2.2%; sugar, 0; fat, 4.7%; fat globules and some very small lymphocytes.	Few Months	Recovery; no final report of case; nature of fluid caused author to abandon idea of lues or peritonitis; the case looks like one of true chylous ascites, due to ruptured lacteal.
5	Kerr	1909	16 D	M	Probably congenital syphilis.	1,140	Milky white, alkaline, odorless, 1,010 to 1,030; profusion of fat cells and a few leukocytes; few cholesteune crystals; chylous urine containing fat and cholesteune crystals.	3	Recovery; the chyluria and chylocele suggest possibility of filaria; Masten, of Mobile, has recorded two such cases in adults.
6	Cowie	1911	7 W*	M	Probably cardio vascular.	2,340	Rich, homogeneous, milky fluid, slightly alkaline, 1,004, no odor; albumin ½ per volume; sugar, strong test; fat, 9.2%; microscopic appearance finely granular; granules in rapid motion; fat, no cellular elements.	5 (7 tappings.)	Recovery complete; no recurrence after two years.
7	Morton	1691	24 M	M	Tubercular glands (bronchial) pressing on thoracic duct near its exit into vein; probably tuberculous.	"Several pounds"	Lactescent chyle, "pure as that found in chyle duct."	12	Died; the fever cause of disease, and autopsy findings point to tuberculosis.
8	Letulle	1885	32 M	M	Congenital cardiac disease.	13,000	Yellow, transparent at first, becoming lactescent later; it was then thicker; Sp. G. 1,009; albumin, 1.2%; fat, 0.015%; no sugar; alkaline.	5 (4 tappings.)	Recovered.
9	Newcomb	1889	24 M		Cause undiscoverable.	?	Chylous fluid, containing lymph cells; no analysis.	?	Recovery (?); a personal communication to Dr. Busey; no further notes obtainable; improved on tappings.

* Doubtless chylous fluid was present in the abdomen as early as the fourteenth day.

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CHARACTERS OF CHYLE AND CHYLIFORM FLUIDS.

Pure chyle as it flows from the thoracic duct is a milky opaque fluid having a slightly alkaline reaction and a specific gravity varying between 1,007 and 1,043, usually not higher than 1,025. It is composed chiefly of fat, proteids, fibrin, extractives and salts and formed elements. These comprise about 10 per cent. of the fluid, the balance being water. Frequently the terms chyle and lymph are used interchangeably. They are not, however, entirely identical, although they are both found in the thoracic duct in comparatively pure state, depending upon conditions under which they are obtained. The principal difference between chyle and lymph lies in the fat content. Chyle may be considered lymph laden with an excess of fat. One may accordingly obtain pure lymph from the fasting animal, chyle from an animal fed particularly on fatty food. The percentage of fat in chyle is therefore an ever-varying quantity, depending upon the character of the food ingested. This fact may be taken advantage of in the differential diagnosis of chylous and chyliform fluids in the serous cavities.

The following table gives the analyses of chyle taken from the thoracic duct:—

Author	Hoppe. Seyler.	Owen. Rees.	Munk.	Hoppe. Seyler.	Hoppe. Seyler.
Animal	Ruptured duct, man.	Executed man.	Man during operation.	Dog.	Horse.
Fat	2.7	0.92	3.3	6.48	0.05
Proteid	3.66	7.08	3.2	2.10	2.98
Fibrin		trace	0.1	0.11	0.12
Salts	0.71	0.4	0.8	0.79	
Other organic Substances ...	0.63	1.8		0.23	0.25
Total solids ...	5.92	9.52	7.8	9.62	0.74
Water	94.07	90.4	92.2	90.67	95.61

The highest per cent. of fat in human chyle, recorded by Munk, is 5 per cent.; in the dog, 15 per cent. It is uniformly emulsified. The individual globules, microscopic in size, resemble

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myriads of micrococci in active Brownian motion. Occasionally the droplets run together to form larger globules. The fat is made up of cholesterin, lecithin, olein, palmatin and stearin, all of which are soluble in ether after first treating with an alkali, and may be recovered from the residue of the etherial extract by appropriate measures. One thousand parts of the dried residue yield in two specimens examined by Zawilski—

	First specimen.	Second specimen.
Cholesterin	113.2	140.9
Lecithin	75.4	88.4
Olein	381.3	770.7
Palmatin	430.1	

The percentage of fat in chyle, chylous and chyliform fluids is conveniently and satisfactorily estimated by the Babcock concentrated sulphuric acid method. For demonstrating cholesterin it is usually necessary to examine the residue of the etherial extract. In the event that a large amount of fat is present this must first be extracted with alcoholic KOH; boiled, and evaporated to dryness, soaps are formed. The residue is now dissolved in water, ether extract made, evaporated to dryness, and the residue redissolved in boiling alcohol, partially evaporated and set aside to crystalize. Typical cholesterin crystals, plates, form and give a red to blue color when treated with a weak solution of sulphuric acid.

The proteids of chyle are chiefly serum albumin and serum globulin. These are coagulated by heat; a drop of HNO_3 brings a yellow color. (Xanthoproteic, or Mulder's reaction.) Fibrin is demonstrated by the ability of the fluid to coagulate. Pure lymph coagulates very rapidly, chyle less rapidly. There is always present in chylous fluids which do not coagulate readily a certain amount of fibrinogen, which will coagulate on addition of some blood serum. In chylous fluids, after several days' standing or in some cases even longer, curd-like masses separate out and rise to the top, leaving a whey-like layer below. These masses, which vary in size up to that of a pea, are sometimes made up of a fibrin network, in the meshes of which are entangled the emulsified fat, etc.

Sugar is a normal constituent of chyle. It is present in the

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same proportion as the sugar of the blood. Sometimes larger quantities may be demonstrated. A chylous fluid must not be considered sugar-free unless Fehling's solution is not reduced after one or two hours, and the fermentation test is negative. There are other substances present in some chyliform fluids which will reduce Fehling's solution.

Microscopic Examination of Chyle.—Through the work of Biedl and Decastello, Selinoff, Crescenze, and others, and more recently of Rous, it has been conclusively shown that the thoracic duct is the chief pathway of the lymphocytes to the blood. These cells, as Rous points out, vary in their number according to whether the animal is at rest or in activity, the number being greater during exertion. Polymorphonuclear cells and red blood cells, if present in any appreciable number, are due to contamination. One must, therefore, not expect to find large numbers of these cells in a pure chylous effusion. He must also not lose sight of the fact that inflammatory affections associated with increase in the leukocytes might also be the cause of a rupture of one of the chyliferous vessels. In a recent text-book the statement is made that there is little use of going into a detailed description of chylous fluids, as they are almost invariably due to carcinoma or some malignant disease of the abdomen.* It has been definitely proven that a milky effusion may be due to a large content of epithelial cells or cells which have undergone fatty degeneration. Cells undergoing mitosis are not normal constituents of pure chyle. In chylous ascites due to constriction of the thoracic duct it would seem probable that the total lymphocyte count of the blood would be relatively decreased and a relative leukocytosis result. A reduction of 79 per cent. was observed by Biedl and Decastello after ligation of the lymphatics on both sides of the neck in dogs.

One must conclude, it would seem, that chylous and chyliform ascites in sucklings is a very rare condition; that instead of regarding it a disease entity it should be considered a symptom-complex dependent upon one of several possible etiologic factors. The etiologic factor to a great extent determines the outcome of the case. Evidently those cases due to some cardio-vascular anomaly or non-inflammatory or cystic condition tend to com-

* This, as well as similar statements found elsewhere, gives me the excuse for incorporating this section on the characters of chylous and chyliform fluids in this report.

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plete recovery, while those due to intrathoracic and intra-abdominal growths, such as tuberculous glands, and to inflammatory conditions tend to dissolution.

The writer wishes to express his thanks to Professor Earle W. Dow for verifying and correcting the French translations, and to Dr. L. F. Warren for the excellent orthodiagram.

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DISCUSSION.

DR. BOVAIRD.—I would like to ask the doctor what were the evidences on which he termed it chylous? There are two different types—one true chyle and the other due to degeneration of the cells in the fluid giving the appearance of chyle.

DR. ADAMS.—Some time ago I assisted Dr. Busey in the construction of his book on "Diseases of the Lymph Channels." I was his student and could present the doctor with a copy of the book. The cases were collected with a great deal of trouble and expense and a considerable amount of research work was done. I am very glad to know that his work is not forgotten.

DR. COWIE (closing): Dr. Busey's work is certainly most delightful. The scholarly manner in which he went about it is very fine.

In answer to Dr. Bovaird, all the analyses are given in the paper, but were not referred to in the presentation. Chylous ascites and chyliform ascites are entirely different things. Chyliform ascites is what we find in chronic peritonitis and carcinoma for instance. In true chylous ascites we must, of course, get the elements of chyle in the fluid. If you starve a dog you can get pure lymph from the thoracic duct; if you feed the dog fat you get chyle. In true chylous ascites, as in this case, we might expect the percentage of fat to vary from time to time.

Another point I wanted to bring out was the importance of blood analysis in this child. I believe that in these cases we should get a relative lymphemia. The lymphocytes get into the blood through the thoracic duct; accordingly if there is an obstruction of the duct it seems reasonable to expect a decrease in lymphocytes in the blood. We might also expect to find increase of lymphocytes in the chylous fluid; unfortunately no blood counts were made in my case; I had to hunt over several spreads made from sediment of the chylous fluid in order to get a hundred cells. These were all small lymphocytes.

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OBITUARIES.

ALOIS MONTI,
JOHANNES THEODOR AUGUST STEFFEN,
HEINRICH VON RANKE,
WILHELM CAMERER.

BY A. JACOBI, M.D., LL.D.

Within a year Germany has lost four eminent pediatrists—not Germany only, but all of us. All their names have been in the front rank of pediatric literature for decades. Their impress on medicine is of more than an ephemeral character. A good deal of what for us is common property was of their making. It is becoming that Americans should be acquainted with their names and doings, and that we should cherish the memories of those who have laid the foundations of pediatrics, such as we enjoy today.

ALOIS MONTI.—Born October 13, 1839, died October 30, 1909. He was the son of a court employee in the provincial service of the country, in Lombardy, and was educated in the Italian college of his neighborhood. Medicine he studied in Vienna; graduated in medicine in 1862, and in surgery in 1863. Professor Mayr—whose name is a deserving one in Austrian pediatrics—taught him the diseases of children, thus enabling him to act as a volunteer assistant in the St. Annen Kinder Spital. There he was soon advanced in rank. In 1869 I met him when he was the enthusiastic and active first assistant of Widerhofer, who was frequently absent in the service of the young imperial family and had often to rely on Monti as his representative in the class-rooms and the hospital wards. Monti began his university teaching in 1870, in the capacity of a "Privat-Docent" of pediatrics. In the year 1887 he was made "Extraordinary" (adjunct) Professor. In 1908 he received a high title, but was never promoted to an "ordinary" regular professorship. That he owned several decorations of his and several other governments is almost self-understood. What

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is more important is that he was a member of learned societies in Rio Janeiro, Brussels, Moscow and Paris.

His contributions to medical science and art are very numerous. They were published in the *Jahrbuch für Kinderkrankheiten*, next in the *Centralzeitung für Kinderheilkunde*—which he founded with Baginsky—and in the *Archiv für Kinderheilkunde*. Some valuable essays of his have been published in Gerhardt's *Handbuch*. Besides many papers on epidemic cholera, renal diseases, spinal hyperemia, tenia, artificial feeding, anemia, etc., he published "Croup and Diphtheria," Vienna, 1879, second edition, 1884, and, together with Berggrün, "The Chronic Anemia of Childhood," Leipzig, 1892. Since 1897 he edited the lectures delivered at the Policlinic, under the title, "Pediatrics in Single Papers" (*Kinderheilkunde in Einzeldarstellingen*); they fill three large volumes, and were followed, in 1906 and 1907, by six supplementary papers on the infections of the oral and pharyngeal cavities, on feeding, on growth, etc.

In 1887 he was one of the founders, and soon became the director, of maritime hospitals and asylums for sick, or scrofulous, or rickety children. They carried long-winded names of people in high positions, even of the sempiternal Austrian Emperor. That is why Monti worked incessantly until he succeeded in transferring the administration of all of them to the city of Vienna as late as 1908. In November of the same year he had his first apoplectic stroke; a new one in April, 1909, incapacitated him.

His practice was immense amongst the rich and aristocratic—who seldom have the intellect to select a good doctor, but still appreciated him on account of his knowledge, efficiency and plain talking—and amongst the poor, whose children he benefited. He was a good man and a great physician, not always pleasant in the presence of ignorance and neglect, and probably careless in regard to professional advancement, but one that has aided pediatrics more than Widerhofer, who was prevented from so doing by his obligations to the reigning family. Monti never lost sight of the scientific accuracy of modern medicine, and altogether exhibited the traits of the great clinicians, whose number is not rapidly increasing during the last few decades.

JOHANNES THEODOR AUGUST STEFFEN was born at Stettin, Germany, December 6, 1825, and died January 7, 1910. He studied medicine at the Universities of Bonn, Heidelberg and

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Halle. At the latter he was graduated in 1848. During the years 1847 and 1848 he was assistant in the medical clinic of Pfeuffer, in Heidelberg, and in 1850 he settled in Stettin, where he practiced general medicine. At an early time he paid particular attention to pediatrics. In 1853 he became the chief physician of the Child's Hospital in his city. That position he retained until 1894. Between 1865 and 1870 he published "Clinic of the Diseases of Children," in which he treated of the lungs and pleura. In 1879 he wrote for Ziemssen's Handbook, Vol. IV., on "Spasm of the Glottis," and on "Whooping-cough"; for Gerhardt's great and epoch-making handbook (Vol. V.) "The Diseases of the Brain." The third volume of his "Clinic," containing "The Diseases of the Heart," appeared in 1889. Many are his contributions in the *Jahrbuch für Kinderkrankheiten*, of which he was one of the editors, and in the "Transactions of the Society for the Diseases of Children," which is one of the most prominent sections of the German Association of Physicians and Naturalists. "Some Important Diseases of Children" appeared in 1895; "The Malignant Tumors of Infancy and Childhood" in the same year.

Heinrich Von Ranke was born May 8, 1830, and died March 12, 1910, at Munich, where he was "Extraordinary" Professor of Pediatrics since his appointment in 1874. He was through a number of years the resident physician of the German Hospital in Dalston, London, in close proximity to, and acquaintance with, Charles West, and from time to time returned to England, with whose ways and literature he was thoroughly conversant. After his return to Munich he became the director and leading spirit of the Hauner Child's Hospital, and organized a large children's dispensary, which, together with the hospital, afforded him ample material for study and practice. Noma, diphtheria, hydrocephalus, spina bifida, and other subjects, were treated by him in special essays and pamphlets. Since his arrival in Munich its university was one of the principal places in Germany where diseases of children were taught and studied, for besides being an expert in his specialty he was known as a man of great general culture, a good teacher and a noble man.

Wilhelm Camerer was born October 17, 1842, and died March 25, 1910. For nearly a decade before his death he was confined to his bed or room by the results of rheumatic carditis,

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but still was always active. The *Monatschrift für Kinderheilkunde,* 1910, No. 1, enumerates his contributions to science and practice under 113 headings. He never wrote a text-book, though there was a time when he was only quite young and another time when he was full of ripe experience. He has earned and deserved his fame without it, and without a professorial position. He always engaged in the practice of medicine in small German towns, in which he worked without complex methods and without the aid of a large laboratory. Still none of the results of his labors have been refuted. They were such as to permit him to apply them to the practice among, and the therapeutics of, children. Pamphlets and essays on disinfection, metabolism, urine, growth of bulk and length, digestion, gout, composition of the young body, woman's milk, psychophysics, and statistics, historic and social questions, have emanated from him and indicate the breadth of his knowledge and the width of his horizon. Evidently the time has not yet passed in which eminence can be realized without prominence. Camerer's temperament and ambition were as modest as his surroundings. Still, he is recognized in Germany, and should be appreciated generally, as one of the makers of modern pedology.

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ADDENDUM

PROCEEDINGS OF THE TWENTY-FIRST SESSION

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PNEUMOTHORAX: A CLINICAL AND RADIOGRAPHIC STUDY.

BY FRANCIS HUBER, M.D.,

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AND

I. S. HIRSCH, M.D.,

Pediatrist to the Jewish Maternity Hospital; Roentgenologist to Beth Israel and Lebanon Hospitals, etc.

Historical.—Pneumothorax as a clinical or pathological entity was not known to the fathers of medicine. They did not differentiate it from empyema; and it was in the diagnosis of this latter disease that they applied the succussion test. This sign—a splashing sound produced in the chest by shaking the patient—was first described in writings credited to Hippocrates and the procedure is, therefore, frequently called "Hippocratic succussion." For over two thousand years, until the time when Laennec made his classical contribution to the subject of pneumothorax, the true significance of the succussion sign was not understood. The presence of the splashing sound was thought to indicate a moderate amount of pus in the chest, while its absence was interpreted as indicating a chest filled with pus.

As time passed, the importance of this sign was questioned, and it finally came to be considered as having but little value. The reason is apparent when it is remembered how infrequent is the occurrence of hydropneumothorax as compared with empyema, and consequently how seldom the characteristic splashing sound was heard. Thus it was that Morgagni[1] stated that "this symptom neither is nor can be perpetual," and he thought it of little value because of its rarity. With the identification of pneumothorax as a distinct entity, the true meaning of the succussion splash was demonstrated, and its value, as a sign of the presence of fluid associated with air, was established.

That pneumothorax remained unrecognized until the nineteenth century is not at all surprising, for it appears that until 1746 it was believed that air was normally present in the pleural cavity. At that time Haller[2] disproved this view, though a long time elapsed before the correct ideas gained general acceptance.

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To judge from the cases reported by Combalusier[3] (1747), Meckel[4] (1759), Ponteau[5] (1783) and Bell[6] (1784) the presence of air in the pleural cavity was at that time recognized as an abnormal condition, and from the proposal of Alexander Monro[7] (1760) and Hewson[8] (1767) to perform thoracentesis for its removal, it is logical to infer that the ill effects of the air in the pleural cavity were understood at that date. Still, no clear or definite idea of the disease existed until 1803, when Itard[9] described the condition as found at autopsy and termed the disease pneumothorax. In a crude way he classified the causes of the affection and recognized its relation to tuberculosis. But neither he nor Bayle,[10] who also described the autopsy findings in a group of cases, was able to recognize the condition during life.

Laennec,[11] however, in 1819, firmly established the disease as a clinical entity. He was perhaps the first to diagnose pneumothorax during life, and he insisted that this could only be done by means of mediate auscultation and the stethoscope. Though his classification is in some respects erroneous, though some of his cases were not true cases of pneumothorax, and though he failed to note some of the accompanying physical changes, his contribution nevertheless stands as one of the milestones of progress in the art of physical diagnosis.

After Laennec's contribution the subject of pneumothorax received considerable attention and discussion. Numerous cases were reported, and the physical signs were investigated and analyzed. According to Emerson, just as Laennec considered mediate auscultation indispensable in the establishment of the diagnosis, so Poirry[12] (1828) maintained the importance of mediate percussion, while Louis[13] (1836) and his pupils laid the greatest emphasis on the clinical history regarding auscultation and percussion as unimportant diagnostic methods. It was not long, however, before these discordant claims were harmonized and the importance and the value of each method duly recognized.

In 1830 Reynaud[14] for the first time collected and reviewed all the literature on the subject and gathered 48 cases. Saussier[15] (1841) wrote an excellent thesis and collected 169 cases. Biach[16] (1880) collected the largest number of cases, 485 from literature and 433 from the records of Vienna hospitals, a total of 918. Drasche[17] (1899) reported and analyzed 198 cases which had

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come under his personal observation during a period of forty years. Emerson [18] (1903) exhaustively reviewed all the literature upon this subject and reported 48 cases from the Johns Hopkins Hospital. To this latter valuable contribution we desire to express our indebtedness for much of the historical data.

It is surprising to note the little attention this subject of pneumothorax receives in the text books on pediatrics. Pneumothorax is not a rare affection in children. It occurs more frequently than is generally supposed. Holt mentions the condition only in its relation to the pathology of tuberculosis. Koplik discusses it briefly in relation to empyema. Netter and Carr merely comment on its rarity. Rotch, Baginsky, Pfaundler and Schlossman make no mention of it.

It is our purpose to review the subject in a general way to record clinical and radiographic observations and then to discuss the condition with reference to its occurrence in children.

Classification.—Pneumothorax may be produced in any one of three ways: First, by the admission of air into the pleura through a penetrating lesion of the chest wall, the type designated as pneumothorax with an external fistula. Second, by the admission of air into the pleura through a lesion of the lung and visceral pleura or through a communication with a gas-containing organ—pneumothorax with internal fistula. Third, by the bacterial decomposition of a pleural exudate with the formation of gas. When, as in this last variety, there is no communication with the exterior air, a closed pneumothorax is said to exist, in contradistinction to the other variety, in which the air has free egress and ingress, and which is designated as open. The sealing of the fistula either by inflammatory activity or otherwise, may convert a primarily open pneumothorax into a closed one. A peculiar formation of the fistula may permit the air to enter the pleural cavity, but prevent its exit. This is the so-called valvular variety. According to the experiments of Marais and Castlenau[21] and Lister,[19] a small wound in the lung is always a valved opening which permits the air to travel in one direction only; that is, from the bronchi to the pleura. This was later emphasized by Bard,[20] who states that it is impossible in a normal lung (one free to retract or collapse) to produce a fistula permeable in both directions; that is, one which will permit the egress and ingress of air into the pleura.

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Causes.—The first variety, according to the classification given, may be produced by stab or gunshot wounds of the chest, by surgical operations, by the accidental admission of air during exploratory puncture and by necrosis of ribs or axillary cellulitis, and, finally, in consequence of the perforation of the chest wall, by a purulent effusion, the so-called "empyema necessitatis." It was recognized early in the study of this condition that not all perforating wounds of the chest will produce pneumothorax, and in Biach's[16] statistics 21 cases with such wounds are cited in which pneumothorax did not result.

The second variety may result from the rupture into the pleural cavity of a tuberculous, purulent or gangrenous focus in the lung, from perforation of a bronchiectatic cavity, or an emphysematous vesicle or from the laceration of the lungs by fractured ribs. It may be produced by compression of, or severe trauma to the chest without evidence of external wound or fracture. Vigorous and prolonged attempts at artificial respiration may give rise to pneumothorax and subcutaneous emphysema. Any cause, in fact, which produces increased intrapulmonary pressure may result in pneumothorax. Thus it has been caused by the severe coughing paroxysms of pertussis and by the straining of persistent vomiting. It has resulted from the perforation of a foreign body in the bronchus. Ulcerative and suppurative processes in the adjacent organs, liver, stomach, esophagus or intestine may perforate into the pleural cavity.

There is a variety of pneumothorax which has been called "spontaneous" because it occurs in apparently normal individuals without evident cause. In these cases the pneumothorax is rarely fatal. Many observers, among them West,[22] deny that the lungs are really normal in such cases and believe that the condition is due either to a latent tuberculosis or to the rupture of emphysematous alveoli. The cases which follow slight exertion, or occur without apparent cause, indicate that in some individuals the lungs because of certain structural peculiarities may be susceptible to rupture. Thus in a case reported by Grenier[98] the pneumothorax recurred three times, in each instance without apparent cause.

Mechanics.—The changes produced in the pleural cavity by the entrance of air, the resulting disturbance in the mechanics of respiration, the position and behavior of the lung under the unusual condition and the modus of the restitution to normal, are

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some of the phases of pneumothorax which have been the subject of much investigation and controversy. These questions are still in many respects unsettled. The conflicting observations recorded by various investigators make it difficult to arrive at any correct conclusions. The lungs may be considered as two hollow, elastic and distensible structures communicating freely with the exterior and suspended within a closed and rigid chamber—the thorax. Each lung is enveloped by a serous membrane, the pleura, which is continuous over the inner surface of the chest. In the normal state the visceral and parietal layers are in contact during expiration and inspiration, so that the pleural cavity, as such, may be said not to exist. Because of its free communication with the exterior, the pressure of the air within the lungs varies but slightly from that of the atmosphere. It is, however, exactly that of the atmosphere only at the end of quiet inspiration and expiration. During the act of inspiration, as the capacity of the thorax is increased, the lungs by virtue of their elasticity, are distended and the intrapulmonary pressure falls. An inrush of air through the trachea takes place until the atmospheric pressure is re-established. During expiration, because of the collapse of the chest and the recoil of the lung, the intrapulmonary pressure is slightly increased over that of the atmosphere, and air escapes. Even during the most forcible expiration, however, the elastic tension of the lung is not satisfied; it is still on the stretch. Between the pleural layers there is a pressure less than that of the atmosphere which is referred to as a negative pressure. This negative pressure varies during the respiratory phases and is greatest at the end of inspiration. It has been estimated by Aaron[23] as varying from minus 4.64 mms. Hg. at the end of quiet inspiration to minus 3.02 at the end of expiration.

The forces concerned in the maintenance of the lung in its normal position may be divided into two classes: Those acting from without, which may be styled extrinsic, and those due to properties inherent in the lung tissue itself, which may be called intrinsic. The extrinsic forces consist of, first, the pressure of the atmosphere on the exterior of the chest; second, the pressure of the air within the lungs (intrapulmonary) which, with the variations noted, is that of the atmosphere; and third, the tension which exists between the pleural layers (intrapleural negative pressure). The intrinsic forces consist of, first, the elasticity of

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the lung, which constantly tends to diminish the tension under which its structure has been placed; and second, the tonus of the lung, due to its bronchi, muscular and vascular tissue, which maintains the definite structure and shape of the organ.

When air is admitted between the pleural layers, the negative intrapleural pressure is diminished; that is to say, it approximates the atmospheric. The lung may now satisfy its inherent tendency to shrink. But under certain circumstances there is not only no shrinkage, but there may be an actual protrusion of the lung through the wound in the chest wall, constituting a hernia. There are evidently conservative forces which counteract the tendency of the lung to retract. The older writers sought to explain this non-collapse by assuming that a state of cohesion existed between the pleural layers. The resistance encountered when an attempt is made to inject air or fluid into the pleura seemed to give force to this idea. Dolbeau and Smith[24] compared this cohesion to that which exists between two opposed wet plates of glass. West[22] believed in this cohesive force and estimated it as being equal to the pressure of 12.5 mms. Hg. But other and later investigators (Northrup[25] and Emerson[18]) have by experiments on animals produced evidence against this cohesion. They have also sought to prove that even the smallest opening of the pleura invariably results in the immediate and complete collapse of the lung, and that under these circumstances the lung takes no part in respiration while the pleural wound is patent. The experiments of others (Hellin[26] and Reinboth[27]) give results which are at variance with these, and demonstrate, on the contrary, that the lungs of animals do not collapse perfectly, even when the pleural opening is the diameter of the trachea, and that the lungs functionate, in the presence of air, in the pleural cavity.

Without attempting to reconcile these antithetical observations, we would insist that it is necessary to be extremely cautious, in the application of the results of animal experimentation in pneumodynamics, to conditions obtaining in human beings; this for several reasons: First, because the anatomical formation of the pleura is different in animals. In the dog, for instance, there is often a communication between the two pleural cavities just above the tendinous centre of the diaphragm, so that nearly every pneumothorax is really bilateral; second, the intrapleural tension is greater in animals; third, the mediastinum is more freely mov-

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able in animals. Sehrwald[28] has shown how the mobility of this structure will affect the condition of both the sound and the affected lung; fourthly, the position of the animal influences the activity of the lung in the affected thorax, and Reinboth[27] has furthermore proven that even in the presence of a large opening in the chest wall, if the animal be so placed that the perforation is lowest, the lung will continue to functionate.

These factors undoubtedly influence the experimental results. In their application to human beings, such results should not be permitted to outweigh the information gained by clinical studies. For whatever the experiments on animals may show to the contrary, the following observations remain indisputable, namely, that in human beings pneumothorax does not always result when a communication is established between the pleura and the outside air, that complete collapse of the lung does not usually follow the admission of air into the pleural cavity, and that finally even when pneumothorax is present the lung in the affected cavity usually continues to functionate to a greater or less extent.

In the explanation of the non-collapse of the lung, it is necessary to take into account besides the factors which influence its position (the extrinsic and intrinsic forces), the power of vicarious distention of its alveoli which the lung tissue may exhibit in response to an afferent impulse. The influence also of the size of the pleural wound is important. The integrity of the lung, however, is of greater consequence as affecting the admission of air into the pleural cavity than is the integrity of the chest wall.

When the wound of the pleura is small, that is to say, less than the diameter of the main bronchus to the lung, the tendency of the lung to collapse is counteracted by the compensatory dilatation of parenchyma, in the immediate vicinity of that portion subjected to the stress of the atmospheric pressure. This increase of the pulmonary volume, associated as it is with increased intrapulmonary tension due to increased respiratory activity, is sufficient not only to maintain the approximation of the pleural layers, but when there is added the expiratory pressure from the sound lung, an actual protrusion of the lung tissue into the parietal wound may take place. One condition, however, must be present in order that this may transpire, and that is, the lung must be uninjured and not diseased.

When the opening into the parietal pleura is a large one, air

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enters the cavity with each inspiration, and the lung shrinks to a corresponding degree. It does not, however, collapse to the same degree as if the wound were in its own tissue. Redistention takes place as soon as the parietal opening becomes smaller through the agency of the same forces as mentioned above, except that the expiratory pressure from the sound lung plays a more important part. The lung continues to take an active part in respiration, but its movement differs from that of the sound lung; that is, it expands with expiration and collapses with inspiraton. As the fistula closes the type of respiration changes, and when the size of the opening becomes less than that of the bronchus the normal type again prevails.

When the lung and pulmonary pleura are injured and there results a pneumothorax with internal fistula, the degree of lung retraction will depend upon the size and form of the perforation, on the size and condition of the bronchus leading to the affected parenchyma and on the condition of the lung. Cases are on record in which perforation of normal lungs did not result in pneumothorax. As a rule, however, despite evidences of reparation, as shown by localized atelectasis by the dilatation of the adjoining parenchyma and by the increased expansion, the air nevertheless escapes into the pleura. As has been pointed out previously, because of the localized atelectasis, the fistula permits the air to enter into the pleura, but prevents its egress, and thus the negative pressure of the pleura is rapidly lowered or changed to a positive one. It is this positive pressure which indirectly gives rise to the oppressive symptoms, but which at the same time effectually aids in the closure of the fistula and thus initiates the reparative process. If, however, because of disease of the lung or the presence of pleural adhesions, the collapse of the lung is prevented, or if the situation of the wound be such as to expose a bronchial opening, then the exit and entrance of air into the pleural cavity is free and restitution to the normal is delayed.

Effect on Lung.—The air entering the pleura may first be confined by adhesions to a particular part of the chest, and only later occupy the entire cavity. This frequently occurs in pneumothorax due to rupture of a tuberculous lung. As the air enters, the lung collapses as a whole toward its root, the force of the positive pressure acting upon every part of the periphery and compressing the lung toward this point. Garland,[29] from ob-

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servations upon dead dogs, claims that the base of the lung retracts first, and only after this part ascends for some distance do the other parts collapse. Experimental pneumothorax, as studied with the Roentgen ray, shows that in the living animal the air rapidly diffuses from its point of entry over the entire pleural

FIG. 1—PNEUMOTHORAX—RIGHT.

(L) Collapsed lung lying about its root.
(A) Pleural cavity containing air—brilliantly illuminated.
(H) Heart displaced to the left.
(N) Normal lung.
(D) Diaphragm. Note its sharp outline on the right side indicating immobility.
(S) Scapula. Note the difference in density of the ribs' shadows of the right and left chests.

cavity, and that this primary retraction of the base does not take place, but that every part collapses simultaneously. The position of a collapsed lung in a pleural cavity free from adhesions is al-

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ways at its root. From this site, if the perforation is small, the lung may be observed to expand from its shrunken condition to take part in the respiratory movements. When, however, the perforation is large and the lung is the seat of disease, the collapse of the lung is usually total and the lung lies against the spinal column as a shrunken mass. Adhesions may permit only part of the lung to collapse, and examination with the Roentgen ray of a recent case will show that during respiratory movements the adherent area expands with inspiration, following the pull of the chest, and shrinks with expiration; it is only with forced expiration, however, that the collapsed portion dilates to any extent. With the healing and closure of the fistula, the affected lobe begins to follow the movements of the chest.

Effect on Diaphragm.—The diaphragm is depressed to a varying degree. Its dome is flattened and its movements are inhibited. In many cases the tone is entirely lost and the diaphragm passively follows the pressure changes in the abdominal cavity, rising with inspiration and sinking with expiration. In cases where massive collapse of the lung persists for some time, the diaphragm may so entirely lose its tone as to sag into the peritoneal cavity. The limitation of the motion of the diaphragm in pneumothorax, as well as in incipient tuberculosis, is not due to any intrinsic affection of this part, but entirely to the loss of the supporting power of the lungs. The loss of tone of the diaphragm results in bulging of the lower chest and widening of the intercostal spaces. This distension is no indication of increased pressure within the pleural cavity, but simply an index of the extent of the paresis of the diaphragm and the intercostal muscles.

Effect on Heart.—The mediastinum and its contents may be considered as a movable body, whose position depends upon the surrounding forces. Any change in the intrapulmonary or intrapleural pressure will disturb the equilibrium and affect its position. When the normal negative pressure is lowered by the admission of air, the heart and great vessels will be displaced in the direction of least resistance, and will thus be pushed away from the affected side. The displacement is greater in children than in adults. The displacement is also greater when the left pleural cavity is involved. In these cases, the heart within the right thorax, the distorted aorta and the stretched blood vessels makes a very striking fluoroscopic picture. The position of the heart

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serves as the best index of the amount of air in the pleural cavity. Recovery can only be considered complete when restitution of this displaced viscus has taken place.

Hydropneumothorax.—A not infrequent complication of simple pneumothorax is the accumulation of fluid within the affected

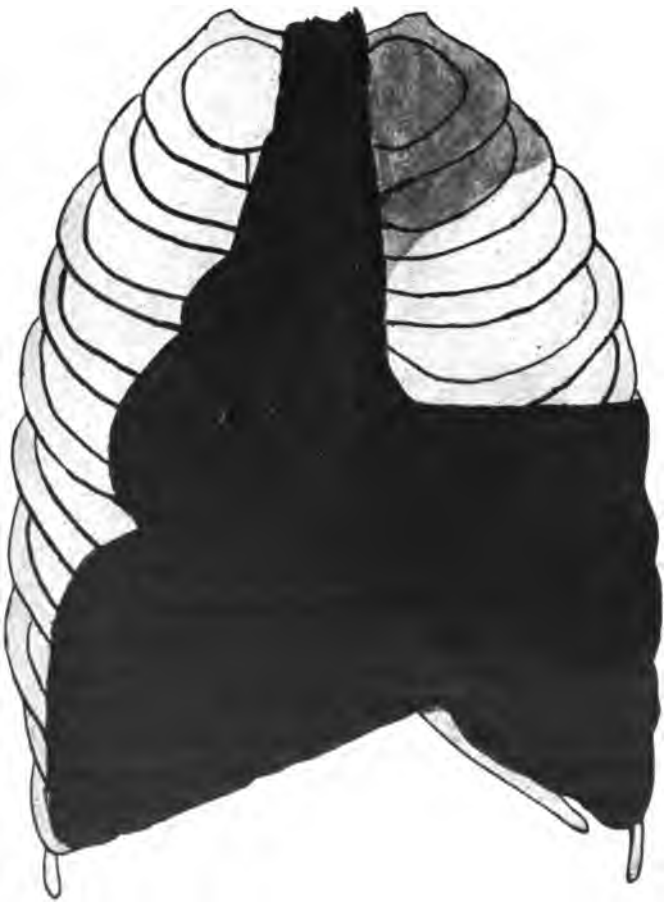

FIG. 2—Diagram of pneumohydrothorax, as shown by Roentgenoscopy. Right side. Upright position. Ventrodorsal view. The three zones are shown. The shaded upper corresponding to the compressed and collapsed lung; the bright middle indicating the air; and the dark lower indicating fluid. This shadow merges with that of the liver below. Heart displaced to the left. The perfectly horizontal line of the upper level of the fluid is shown.

pleura. It has been estimated that but 10 per cent. of all cases are free from this complication. The development of an effusion depends to a great extent upon the condition of the pleura and

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lung prior to the admission of air. If these organs are the seat of an inflammatory condition, the occurrence of an effusion is to be expected, while penumothorax in a previously healthy pleura may go on to restitution without effusion. According to West, the fluid is serous, sero-purulent and purulent in an equable division of cases. In children, purulent effusion is a very common occurrence. It is unusual for a serous effusion to change gradually to a purulent one. The organisms in the fluid are usually those associated with the primary disease. In perforating wounds the infecting organism may be introduced from without. In the case of May and Bebhard[30] the infecting organism was the colon bacillus introduced through a stab wound.

When fluid accumulates in a pleural cavity containing air, the physical conditions, as viewed by means of the Roentgen ray, are different from those of simple effusion. In ordinary exudative pleurisy the upper level of the fluid appears concave, rising higher at the sides and more so at the outer than the inner, while a change in the position of the thorax has but little effect on this level. In the presence of air, however, the upper surface is horizontal (Fig. 2); the level changes freely with the position of the thorax, but always remains horizontal. Moreover, the fluid is affected by every movement of the body. Thus the rise and fall of the level corresponding to the respiratory movements, the undulations synchronous with the systole and diastole of the heart, the agitation of its surface when the chest is percussed or during coughing, and the waves produced by the more forcible succussion are all phenomena to be observed with the fluoroscopic screen. When moderate in amount the level of the effusion rises with inspiration and falls with expiration—a reversal of what should normally occur. This paradoxical condition is due to the loss of tone of the diaphragm, whose movements become altogether passive and follow the abdominal pressure changes. In the same way as the abdominal wall moves forward with inspiration, so the diaphragm is pushed upward in that phase. When the fluid is considerable the diaphragm may become inverted and so weighed down that the level of the fluid is not appreciably affected by the respiratory movements.

In the presence of air the upper limit of dullness does not indicate the level of the fluid. The low tone of the note is vitiated by the tympanitic resonance due to the presence of air. It has

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been stated as axiomatic that in these cases the amount of fluid will be double that indicated by the line of dullness (Skoda[31]). Thus in a case of Senator's, Weil[32] states that the chest was hyperresonant to the twelfth rib even after 800 c.c. of a medicated solution had been added to the fluid already present. A small amount of effusion cannot be made out by the usual methods of examination. The predominating pathological condition will determine the signs which it is possible to elicit. Thus in the presence of considerable gas even a moderate effusion may escape detection, while a small amount of air associated with much effusion will but rarely be diagnosed. The Roentgen ray examination will render considerable aid in these cases, for even the least effusion in the costodiaphragmatic sinus may easily be made out either by the fluorescent screen or on the photographic plate. (Fig. 3.)

Subcutaneous Emphysema.—Subcutaneous emphysema may be a complication of pneumothorax due to exploratory puncture, incision of the chest wall or trauma with or without evidence of fracture or rib. The emphysema may in some instances appear in the tissues of the neck before the air gives evidence of having entered the pleural cavity. This is brought about by the extravasation of air beneath the temporarily intact visceral pleura. From there the air dissects its way into the mediastinal tissues and then into the neck. Later the pressure may cause perforation of the visceral pleura. Such were the events in a case reported by Steffen[79] resulting from the fracture of a rib. Before the importance of pneumothorax was recognized, subcutaneous emphysema interested the early observers, who ascribed the symptoms of the former disease to its complement or complication subcutaneous emphysema. Ambroise Pare[33] (1628) was perhaps the first who recorded observations upon the presence of air in the subcutaneous tissues, resulting from fractured ribs. As a rule, emphysema, when a complication of pneumothorax, does not add to the gravity of the case. Perrin[34] directs attention to the fact that subcutaneous emphysema, following puncture of a pneumothorax, may be a serious complication. The emphysema may become general and require incision or a cellulitis may be set up, if a pleurisy be present. The prognosis, however, is not always grave, and 2 cases are reported in which recovery took place. In both, the emphysema was produced accidentally in thoracentesis. The puncture

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wound acted as a safety valve compensating for the continual accumulation of air in the pleural cavity, by evacuating it into the cellular tissues, where it was absorbed. In a case reported by Bovaird[40] the chest of a child of three years, with left pneumothorax, was punctured. Subcutaneous emphysema followed, which spread over the entire body. It is interesting to note that

FIG. 3—PYOPNEUMOTHORAX—RIGHT. (VENTRODORSAL VIEW.) RECUMBENT POSITION.

(AE) Air and fluid in the right pleural cavity.
(L) Shadow of collapsed lung faintly visible owing to the superimposed shadow of fluid which, because of the recumbent posture, flows over the posterior wall of the chest while the air rises above it.
(H) Heart displaced to the left.
(NL) Lung on the left side in a state of compensatory emphysema.
(D) Diaphragm.
(C) Costo-diaphragmatic space of right side clearly outlined. Compared with appearance of space on diseased side which contains fluid.

the air in the subcutaneous tissues is absorbed more rapidly than in the pleura. It requires from three to seven days for the air to disappear from beneath the skin, while in the chest it may persist for three or four weeks, or even longer.

Composition of Air in Pneumothorax.—The chemical compo-

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sition of the air in the pleural cavity may become changed in consequence of secondary infection or absorption of some of its constituents. The composition of the gas in cases due to the decomposition of the pleural exudate will depend upon the nature of the infecting organism. The most frequent infecting germs are the bacillus coli communis and the bacillus ærogenes capsulatus. In the recorded cases in which the development of gas from decomposition of fluid was supposed to have taken place, paracentesis had been performed, and it is not improbable that the organism may have been introduced in this manner.

Absorption of Air.—That the pleura possesses the power of absorbing gases has been proven by animal experiments and observations on human beings. J. Davy[85] (1823) was one of the first to experiment with the absorptive power of the pleura. Pure oxygen is absorbed more readily than nitrogen, while CO_2 and O were absorbed at about the same rate as air. The relative quota of CO_2, in the air which enters the pleura, becomes rapidly greater than that of the atmosphere. More CO_2 is also present if there is an accompanying purulent effusion. The absorption is retarded in the presence of any inflammatory condition of the pleura or lungs. The air disappears more rapidly in children than in adults. The rapidity of absorption varies within wide limits, though it is not unusual for it to be completed in from four to eight weeks. Fowler and Godlie[86] state that a quantity of air sufficient to give a tympanitic note over half of the chest completely disappeared in three days. In a case reported by Aron[23] of a man with emphysema, who developed pneumothorax from inhaling hydrochloric acid fumes, the gas was absorbed in four weeks. In some of the reported cases it has, however, taken months and years before absorption was complete.

Pneumothorax as a Therapeutic Measure.—Since Houghton's observations (1832) of the apparently favorable influence of pneumothorax upon the progress of pulmonary tuberculosis, this phase of the subject has been a matter of much controversy. Such excellent clinicians as Stokes, Saussier, West and Walshe have failed to observe any distinct definite or lasting improvement. Potain[87] (1888) was the first to recommend the introduction of sterile air after the removal of the exudate. He believed that this immobilized the lung and favored cicatrization and cure. Forlanini[88] (1894) produced pneumothorax in 2 tuberculous cases with

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nitrogen gas, and claimed satisfactory results. Murphy[89] recommended this procedure in unilateral tubercular involvement.

Pneumothorax in Children.—Pneumothorax, though quite a rare disease in children under three years, is not rare in older children. It may occur in the newborn as a result of trauma. Thus Ruge[38] reported the case of an infant who was born asphyxiated. After resuscitation, it cried lustily during the night, but the next morning became cyanotic and died. The autopsy showed left pneumothorax, the pleural cavity also containing a little blood. It was supposed that there had been a rupture of the alveoli, interstitial emphysema, and finally, rupture of the pleura, and that the condition was due to the violent crying, but from similar cases, which have come under our observation, it appears reasonable to suppose that the efforts at resuscitation played an important part in its production.

James[39] refers to a case reported by Money which occurred in a child of three months. Bovaird[40] reported the autopsy record of a case of pneumothorax due to rupture of a small abscess in a child of four months.

Etiology.—In the order of their frequency, the causes of pneumothorax may be stated as follows: Tuberculosis, gangrene, trauma, empyema, foreign bodies and abscess. Pneumothorax frequently complicates those infectious diseases in which bronchopneumonia is a prominent feature, namely, measles, pertussis and diphtheria. It is but rarely due to suppurative processes in the stomach, esophagus, liver or vertebra. The accompanying and basic pulmonary condition is usually a secondary bronchopneumonia. Measles, bronchopneumonia and pneumothorax is a frequent example of the march of events. The perforation may be due to the rupture of a cavity, a purulent process, an emphysematous bleb or to the softening of an infarct.

Tuberculosis.—While in adults at least 80 per cent. of the cases of pneumothorax are due to tuberculosis, in children not more than 40 per cent. are due to this cause. Of this 40 per cent., the vast majority of the patients are beyond the age of four years. It has been estimated that about 1 per cent. of the cases of pulmonary tuberculosis in children develop this complication. Of the 35 cases reported by Lentz,[41] 14 were due to this disease; and of the 31 cases collected by Chrzanewska,[51] 12 were due to tuberculosis.

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One of the earliest recorded instances of tuberculous pneumothorax in a child was reported by Leas.[42] The patient was a girl, two years old, and the pneumothorax was due to the rupture of a tuberculous cavity of the left lung. Sevestre reported a case in an infant of sixteen months due to rupture of a tuberculous cavity. Pollock[43] reported a case due to a similar condition in a child of three and one-half years, in whom tuberculosis had not been suspected. Parks,[45] West,[44] Nichols,[48] Rilliet and Barthez,[46] Seiffert,[47] Gailliard,[49] Revilliod,[69] Steffen,[73] Cozzolino,[50] and Leconte[90] have recorded cases due to tuberculosis. The perforation usually occurs during the active stage of the disease and may be due either to the rupture of a caseous deposit or a cavity. The latter process is but rarely found in infants. The perforation is not always found within the area of the most extensive disease, and is seldom found at the apex. This is due to the protective influence of adhesions. The left side is more frequently involved, and the perforation is found more often in the lower lobe than in the upper. The perforation is, as a rule, very small and difficult of detection. Tuberculous pneumothorax usually terminates in a pyopneumothorax.

Gangrene.—Gangrene of the lung is more frequently a cause of pneumothorax in children than in adults. The gangrene may be secondary to pneumonia, bronchiectasis or thrombosis. Lentz[41] mentions 11 cases due to this condition in a series of 35 cases of pneumothorax. Zuppinger[52] reported 3 cases of pneumothorax due to gangrene of an infarcted area in the course of diphtheria.

Traumatic Pneumothorax.—That pneumothorax may result from trauma of the chest without any evidence of fracture was recognized as early as 1757 by Hewson.[8] Traube[54] was among the first to make a contribution to this subject. It has been suggested that in these cases the injury is produced by a greenstick fracture of the rib, which rights itself when the pressure of the traumatic force is removed. This explanation probably applies only to children. It is also well known that an increase in the intrapulmonary pressure, such as may occur in compression of the chest when the glottis is closed, may result in rupture of the lung and pulmonary pleura. Many such cases have been reported in children. Hematothorax is not infrequently present in these cases. Among the cases recorded in which pneumothorax was produced by trauma without fracture or wound of chest are the following:

Curling[55]: Boy, thirteen years, was run over. Left pneumo-

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thorax resulted. No fracture or external injury could be found.

Butlin[56]: Child, six years, was run over. Left pneumothorax resulted and recovery took place in six weeks. No fracture or external injury was found.

Gould[57]: Child, six years, was run over. Left penumothorax developed two days later. Chest returned to normal one week after aspiration of air. No fracture or external injury found.

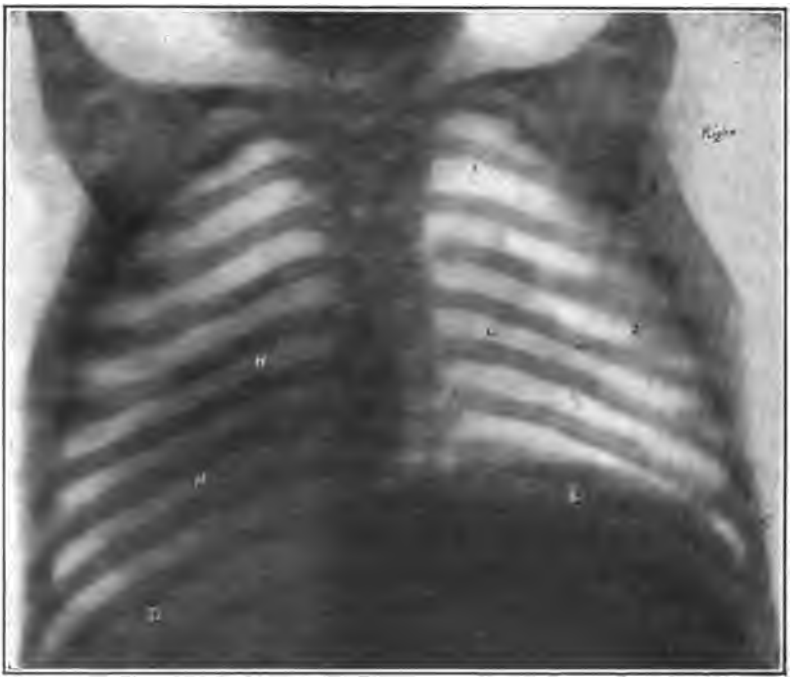

FIG. 4—TRAUMATIC PNEUMOTHORAX.

(L) Partially collapsed lung lying at its root in the middle and posterior part of the right chest. Its three lobes can be made out. The upper extending to the fifth rib, the middle between fifth and sixth, and the lower from this point to the ninth. The arrows are one below and one above the middle lobe.
(A) Air in the right pleural cavity surrounding the lung.
(C) Costo-diaphragmatic space clear, showing no effusion.
(H) Heart displaced considerably to the left. The right and posterior border is seen at the mid-vertebral line. The portion of this right heart percussable was therefore to the right of the sternum.
(D) Diaphragm.
(S) Scapula.

Lucas[58]: Boy, two years, after trauma developed right pneumothorax. Recovery took place in twenty-six days. There was no evidence of fracture.

The following case, which we were permitted to study through

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the courtesy of Dr. Henry M. Silver (Attending Surgeon to Beth Israel Hospital), belongs to this category:—

Hyman T., native of United States, four and one-half years old, was admitted to Beth Israel Hospital, November 27, 1907. About 4 P.M. of the day of admission he was run over by a truck. He was picked up in a fainting condition. On regaining consciousness he expectorated and vomited some blood. There was no hemorrhage from either nose or ears. There were no evidences of pain. The general condition of the child was bad. There was marked dyspnea, a feeble, thready pulse, the skin and mucous membranes were cyanotic, the peripheral circulation was poor and there was considerable shock. At 9 P.M., pulse 142, respiration 62, temperature, 100.4°. Examination showed some slight discoloration of the skin over the right anterior chest, but failed to disclose any wound or any fracture of the ribs. The right chest gave all the signs of pneumothorax. The heart was considerably displaced to the left and its impulse was very feeble. Fluoroscopic examination showed the lung lying in a partially collapsed state at its root. It was motionless, except during forced expiration or coughing, when it was seen to dilate to a moderate extent. The diaphragm rose during inspiration and sank with expiration and its movement was sluggish.

For a week the condition of the child was precarious. The pulse varied from 112 to 190. The temperature was moderately elevated and fluctuated irregularly. During the second week the patient began to rally and the improvement henceforth was progressive. Ten days after the accident fluoroscopic examination showed a condition similar to that in Fig. 4. The contracted lung was seen in the middle and posterior part of the right chest, surrounded by air. During inspiration the lung could now be seen to expand from its partially shrunken state and to fill the chest cavity almost completely. With expiration, it would collapse rapidly to its root, the shrinking being of equal extent in all parts. The diaphragm on the affected side moved slightly. No fluid was visible. The patient was discharged three weeks after admission in excellent condition, with no respiratory difficulty; nevertheless, the radiograph at this time showed that there still was some air present in the affected cavity, and that the heart had not yet returned to its normal position.

Paracentesis.—The production of pneumothorax during paracentesis or exploratory puncture merits consideration since it oc-

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curs more commonly than is generally supposed. Because of the absence of severe symptoms and the presence of but few physical signs, the condition is frequently overlooked. The accidental introduction of air in this way, provided no infection takes place, does not usually add to the gravity of the case, but it is not always as Stokes puts it, "a matter of trivial moment." In an appreciable number of cases it has contributed to a fatal termination. Seven deaths due to exploratory puncture of the chest are reported in the *British Journal of Children's Diseases.*[53] The commonest ways in which air is thus admitted are, either through faulty technique, through injury of the lung by the point of the needle or by leakage of air through the apparatus. The air usually enters from the exterior only after some of the fluid has been withdrawn. Fluids are incompressible, and it is therefore impossible to inject air into a chest already filled with fluid. Sears[59] reported 4 cases of pneumothorax due to paracentesis. In 3 of the cases there were no resulting bad effects, but in 1 the complication undoubtedly contributed to a fatal termination. Gittings[61] reported 3 cases with no untoward results. In one of the cases only a localized pneumothorax followed, but subcutaneous emphysema developed in all. Variot and Roy[62] reported the case of a girl of eleven years, who was tapped for pleural exudate. Immediately after tapping she developed pneumothorax. This was ascribed to rupture of the lung by pleural adhesions, but in all probability it was due directly to the trauma of paracentesis. Packard[64] reported a fatal case of pneumothorax due to perforation of an emphysematous bleb by the point of an aspirating needle. Emerson[18] reported 10 cases due to paracentesis with five deaths. Northrup[60] reported the case of a child of two years, who was admitted to the hospital suffering from severe dyspnea and cyanosis. It was operated on for empyema, but only air escaped when the pleural cavity was opened. The pneumothorax was believed to be due to paracentesis performed before admission to the hospital.

A case of pyopneumothorax, which we believe was due to exploratory puncture, was admitted to the Children's Service of Beth Israel Hospital on September 27, 1909. The child was five years old and had had a mild attack of pertussis before the onset of the present disease. This began three weeks before admission, with fever, cough, rapid breathing and prostration. The fever was higher in the evening, and soon after the onset of the disease was associated with profuse sweats. Three days be-

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fore admission the condition grew worse, the prostration became more marked, there was evidence of pain in the right chest, the temperature fluctuated between normal and 104° and the dyspnea was severe. Before admission a puncture of the chest had been

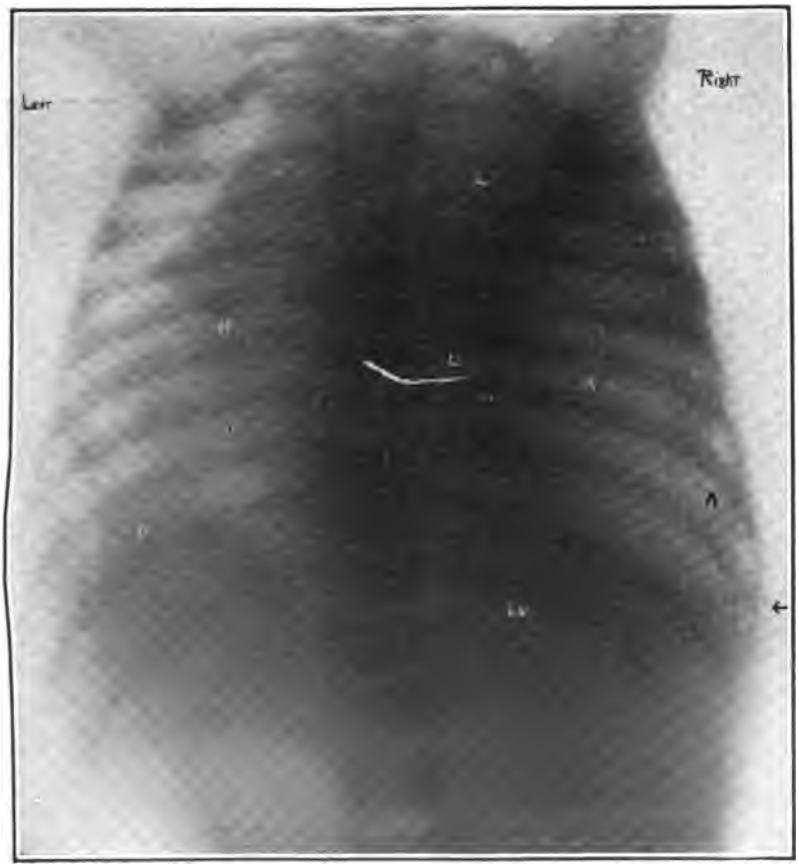

FIG. 5—PYOPNEUMOTHORAX RIGHT. VENTRODORSAL EXPOSURE. RECUMBENT POSTURE.

(L) Collapsed lung covered by fluid.
(AE) Pleural cavity containing both air and effusion.
(A) Air bubble visible in the shadow of the antero-lateral part of the chest.
(H) Heart displaced far into left chest.
(D) Diaphragm.
(Lv) Shadow of liver merging with that of the effusion. The arrow indicates the costo-diaphragmatic space filled with fluid.

made and turbid fluid withdrawn. Examination on admission showed evidence of fluid and air in the right chest. (Fig 5.) Examination of pus obtained by puncture failed to show tubercle bacilli. The Moro and Von Pirquet tests were negative. The ex-

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amination of the sputum did not show any tubercle bacilli. Ten days after admission thoracotomy was performed. Both air and pus escaped. From that time on the condition of the child gradually improved and convalescence is now progressive.*

Besides this case we have seen many others due to paracentesis and some complicated by subcutaneous emphysema. Among them was a child, three years old, seen by Dr. Huber in consulta-

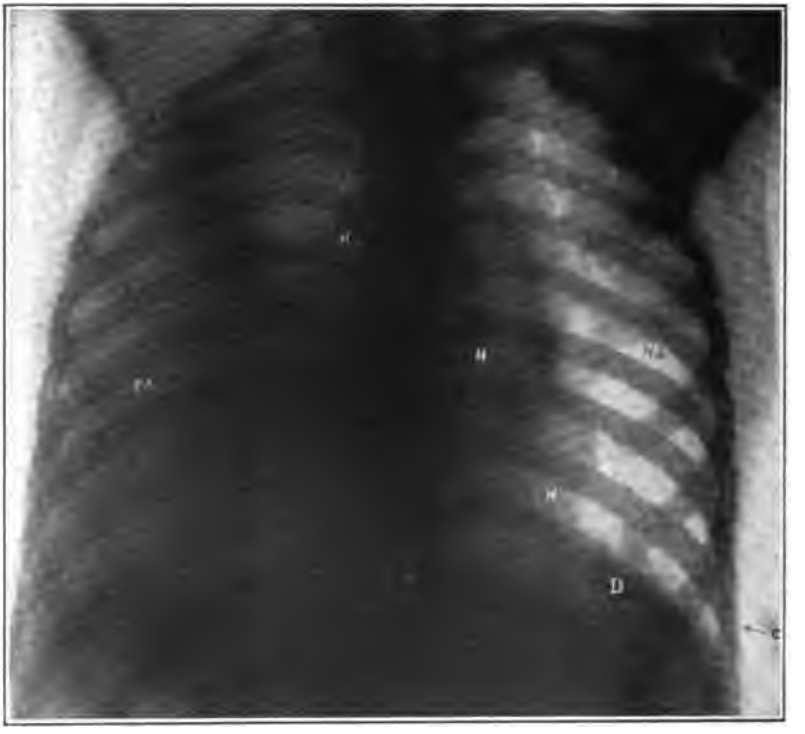

FIG. 6—PYOPNEUMOTHORAX LEFT DUE TO PARACENTESIS. VENTRODORSAL EXPOSURE. PRONE POSTURE.

(EA) Pleural cavity containing air and effusion. Note the lighter areas of the lateral part of the chest due to the air.
(H) Heart displaced to the right.
(D) Diaphragm. On the right side it is at the level of the tenth rib, while on the left its shadow merges with that of the effusion.
(C) Costo-diaphragmatic space of normal side. Compare the appearance of this space with that on the affected side which contains fluid.
(NL) Normal lung, the seat of a compensatory emphysema.
(S) Scapula.

tion. Exploratory puncture of the left chest had been done by the attending physician on the preceding day, because of marked dullness and feeble breathing. This procedure was followed by a pneumothorax and extensive emphysema of the back and lateral

* Since above was written, the child has been discharged cured.

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parts of the chest. A careful examination revealed, furthermore, the physical signs of an effusion on the opposite side, namely, in the right pleura. Exploratory puncture establishing the presence of pus, the right pleural cavity was incised and drained. The emphysema of the left chest wall disappeared in a few days without evidence of infection. The subsequent course, in spite of the pneumothorax on the left side, was uneventful.

Empyema.—Pneumothorax produced by the perforation of a purulent effusion into the lung is not common. Such perforation occurs in the severe cases associated with gangrene of the inflammatory products and in the neglected cases. Tuberculous empyema has a tendency to follow this course. The presence of the air in the thorax may not become apparent for some time after the rupture has taken place, and even then the characteristic signs are not clearly obtained. At intervals, after severe coughing paroxysms, large quantities of foul-smelling pus may be expectorated. Comby[65] reported the case of a boy of eleven years, who developed pneumothorax three days after a purulent effusion in the left chest was noted. The perforation was found to have been produced by gangrenous degeneration of the pleura. Seifert[47] has reported a case in a boy of eight years due to rupture of a left empyema into the lung.

Foreign Bodies.—Direct perforation of the lung by a foreign body aspirated into a bronchus is rare. As a rule, the perforation is caused by a secondary abscess. This complication usually occurs a long time after the foreign substance has entered the bronchus, but in exceptional cases the interval may be brief. Ast's[70] case is remarkable in this respect. The patient was a girl of four years, who had aspirated a spice into the right bronchus. Bronchopneumonia, with severe pleuritic involvement, followed, and on the eleventh day, pneumothorax. In a case reported by Zuppinger[52] of a child of two and one-half years, total right pneumothorax took place during sleep. Death followed in thirty-six hours. At autopsy the perforation was found to be due to a grain of corn which had perforated into the pleura. The foreign substance had evidently been aspirated some time before, but no history of such an occurrence had been obtained. In a case reported by Carslaw[78] the pneumothorax resulted from the perforation of the pleura by a plum stone aspirated into the right bronchus. Foreign bodies impacted in the esophagus have been known to perforate the pleura. Rosenthal[71] reported a case of pneumothorax

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due to the perforation of a bone impacted in the esophagus of a boy. In Müller's case, pneumothorax was produced by the perforation of the left pleura by round worms from the stomach.

Abscess.—Strange as it may seem, pneumothorax due to the

FIG. 7—PYOPNEUMOTHORAX LEFT. VENTRODORSAL EXPOSURE. RECUMBENT POSTURE.

(L) Collapsed and consolidated lung adherent to chest wall at point indicated by arrow.
(A) Pleural cavity containing air and effusion. The greater part of this effusion has gravitated toward the upper part of the affected chest.
(LH) Left heart. The heart is moderately displaced to the right.
(RH) Right heart.
(LD) Left diaphragm at level of eleventh rib posteriorly.
(RD) Right diaphragm at level of tenth rib posteriorly.

rupture of an abscess of the lung is a rare condition. Of Biach's 715 cases, but 10 were due to this cause. Saussier[15] cites 1 case, while Widal refers to 2. Gerard[74] cites a case of Trousseau's,

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and states that this form usually occurs after pneumonia in children or old people. Villey[63] believes the condition to be more common in children than in adults. Emerson[18] cites 2 cases due to metastatic abscess of the lung. In our cases the small abscess was associated with a pneumonia and empyema.

Pyopneumothorax due to rupture of an abscess of the lower left lobe into an empyema. Pneumonia of the lower left lobe. Death.

B. K., seven months old, was admitted to Beth Israel Hospital May 19, 1909, with the history of having been ill for five days with fever, cough, dyspnea and restlessness. Physical examination revealed the signs of an effusion in the left pleural cavity. On admission her pulse was 148, respiration 52, temperature 102.6°. Exploratory puncture showed turbid serum. The cytological examination of the specimen of fluid withdrawn gave 60 per cent. polynuclears and 40 per cent. mononuclears. Pneumococci were present. Because of the presence of an extensive pneumonia, it was considered best to delay operation. From this time until the date of operation, the temperature ranged from 102° to 103°, on two occasions falling to 100°, and once rising to 104° F. The respiration varied between 30 and 50, and toward the end of the week rose to 64. The pulse rate fluctuated between 120 and 140, the frequency, however, gradually rising so that at the end of the week it was 170.

A white blood count, four days after admission, showed 48,000 leucocytes, of which 82 per cent. were polynuclears and 18 per cent. mononuclears. Six days after admission, on the eleventh day of the disease, vomiting and eructation appeared, which persisted to the time of death. Examination at this time showed an increase in fluid, and an exploratory puncture revealed pus. For about eight hours the condition remained unchanged, and then suddenly grew worse. Examination now showed the presence of air and fluid in the left pleural cavity. (Fig. 7.) In spite of all treatment the vomiting and eructation grew worse. Simple incision and drainage of the chest failed to give any relief. The day following the operation the blood examination showed 42,000 white blood cells, with no appreciable change in the differential count from the last examination. The temperature vacillated between 97° and 104° F., pulse varied from 120 to 170, and respiration between 34 and 60. The condition gradually grew worse, the restlessness was marked, the vomiting and eructation were persist-

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ent, muscular twitchings appeared, and eleven days after admission (sixteen days after the onset) the child died.

Only a limited autopsy was allowed. The empyema cavity was found fairly large, and apparently walled off by adhesions above on the level of the fourth dorsal spine and anteriorly about the anterior axillary line. The cavity was traversed by a few bands. The left lung showed marked consolidation of the lower lobe. About midway between the fissure and the lower border, and one inch from the posterior border, a cavity was detected, 1 cm. in diameter. The edges of the cavity were smooth; it communicated freely with the empyema cavity and was lined by pyogenic membrane, $^1/_{16}$ of an inch thick.

From these findings it is evident that a small abscess of the lung had set up a suppurative pleurisy, limited at first, but gradually extending, and finally, in consequence of the intractable vomiting, the adhesions had given way with the production of the pneumothorax. The exploratory puncture had been made in the usual manner, posteriorly and below the angle of the scapula, and could not in any way have caused the perforation. Previous to the autopsy, the suspicion may have existed that the pneumothorax was due to the second exploratory puncture. The postmortem examination, however, proved this suspicion to be unfounded. The wall of the abscess cavity was not a recent one, the granulation tissue being of considerable thickness. The pleural cavity did not communicate with the lung through a narrow fistula, but directly by a wide opening. These conditions are not found in pneumothorax due to paracentesis. On the other hand, the autopsy showed that the abscess was a superficial one, associated with a pneumonic process and accompanied by a purulent effusion. The empyema was sacculated, as it very often is in infants, and the exudate contained considerable fibrin, a condition usually present in pneumococcic infections. These factors would have been sufficient under ordinary circumstances to minimize the possibility of the rupture of the abscess had not the strain of the persistent vomiting acted as an excitant.

Bovaird[40] reported the autopsy findings in 2 similar cases. In 1 case the child was four months old, and the right pleural cavity contained, in addition to the air, about 1½ ounces of thin muddy fluid. The right lung was compressed, and in its lower lobe were found two small abscess cavities about 1 cm. in diameter. One of these had perforated the pleura and produced the pneumothorax.

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In the other case the infant was six months old. There was left pneumothorax, and in the left upper lobe there were scattered pneumonic areas. On the anterior surface of this lobe a thin walled abscess cavity had ruptured into the pleura. The lung about this abscess was consolidated and compressed.

Pertussis.—Aside from the bronchopneumonia, which is a frequent and serious complication of pertussis, vesicular emphysema is present in all severe cases. As in diphtheria, these complications predispose to the production of pneumothorax, only to a greater degree. It mostly occurs, however, in debilitated children, and the process is often hastened by the combination of an inflammatory condition superimposed upon the already weakened and emphysematous lung. Thus in a case reported by Winocouroff[75] a child, four years old, after suffering from pertussis for one month, developed a croupous pneumonia, and on the third day of this disease, after a severe attack of coughing, developed pneumothorax. In Malinowski's[76] case the pneumothorax followed a paroxysm of coughing, excited by compression of the chest. In von Gelmo's[77] case the pneumothorax was accompanied by subcutaneous emphysema. In a case reported by Villy,[78] a child, three years old, convalescing from diphtheria, developed pertussis and very soon after a pneumothorax.

Diphtheria.—Diphtheria is a disease in which pneumothorax is not an infrequent complication or sequel. There are three complications which are rather frequent in diphtheria. The first is bronchopneumonia, which is usually of the severe type; the second is emphysema, vesicular or interstitial, and this is more common if the larynx is affected; the third is thrombosis or embolism, which may affect the vessels of the lung. These pathological conditions all predispose to the production of pneumothorax. Zuppinger[52] reports several cases in which the sequence was diphtheria, infarct, gangrene and pneumothorax. Steffen[79] reported 2 cases complicating diphtheria: one in a child of one and one-half years was due to rupture of an emphysematous lung, and the other, in a child of five years, to softening of an infarct. Schwalbe's[80] case was a diphtheric child, who developed subcutaneous emphysema and double pneumothorax six days after a successful tracheotomy had been done. Torday[81] reported a case which followed laryngeal diphtheria. Cnopf[82] reported 4 such cases during the course of laryngeal diphtheria.

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Measles.—The bronchopneumonia complicating measles is usually severe and may have associated with it the pathological conditions which predispose to rupture of the lung, namely, emphysema, gangrene and abscess. Zuppinger speaks of the frequency of pneumothorax during the course of severe measles. Steffen reported a case in a girl of seven years due to rupture of a small abscess. Bovaird reported 2 cases occurring during the course of measles. One was a child of four years, who, on the ninth day of measles and bronchopneumonia, went into collapse suddenly with cyanosis and dyspnea. The examination revealed a right pneumothorax. At autopsy the pleura contained about an ounce of pus, the lung was compressed and consolidated and contained bronchiectatic cavities filled with pus. The other patient was a boy of three, the pneumothorax developed on the eighteenth day of the measles. Following an exploratory puncture, subcutaneous emphysema developed, which spread over trunk and arms. At autopsy a perforation was found in the lower left lobe.

Symptoms.—At the instant of the occurrence of the perforation, there is severe pain with evidence of respiratory and circulatory disturbances and shock. Elsberg[84] has proved experimentally that these symptoms are in great part due to the displacement of the mediastinal contents, which lose their support through disturbance of the equilibrium of the air pressure within the thorax. In children these symptoms are unusually severe. This is due to the mobility of their mediastinal structures. The onset is usually sudden, with severe pain, dyspnea, cyanosis and shock. As the viscera accommodate themselves to the changed conditions within the thorax the alarming symptoms subside.

It is surprising to note how quickly in many cases, particularly in older children and adults, a tolerance to the presence of air in the pleural cavity is established. Even with one lung compressed by fluid and the heart displaced, these patients are often comparatively comfortable and have but little dyspnea. This has ever been a source of wonder to clinicians. Houghton[85] in 1832 reported the case of a laborer with left pyopneumothorax, who went cheerfully about his vocation, though the succussion splash from his chest could be heard as he walked downstairs. Adams[86] reported a case in which the pneumothorax, which resulted from a slight strain, persisted without exudate or infection for a period of four years without any marked inconvenience.

Physical Examination.—The child, as a rule, prefers to lie on

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the affected side. When the dyspnea is severe, the activity of the accessory respiratory muscles is evident. Inspection will show that one side of the chest is more or less immobile and distended. The lower ribs flare out, the intercostal spaces are widened and the depressions obliterated. The hypochondriac region corresponding to the affected side is prominent.

The cardiac impulse is displaced, and, if the left chest be affected, the heart impulse may be seen to the right of the sternum, even as far as the mammary line, and distinct epigastric pulsation will be noted.

The information gained by inspection is not characteristic of pneumothorax, since simple effusion often gives a similar picture.

Tactile fremitus is a sign not easily obtained in older children. It will, however, usually be found absent or diminished in intensity.

Percussion must be gentle, for the air in the chest makes it a sounding-board which readily exaggerates and transforms every sound and obliterates the finer differences of tone. With the exception of an area in the upper intrascapular region, the entire chest may be tympanitic from apex to base. This note will also be obtained below the level at which pulmonary resonance may be elicited normally. This is a valuable sign, and in doubtful cases will be of great service. Less frequently the note may be either hyperresonant or normal over the entire chest. There is usually an area of normal or relatively dull resonance in the intrascapular region. This is due to the more or less collapsed lung lying at its root. When the pneumothorax is due to phthisis, the apex may be adherent, and then a normal or relatively dull note will also be obtained over the upper part of the chest. Infrequently a cracked-pot sound may be elicited. The absolutely flat note generally associated with effusion in the pleural cavity is seldom obtained when the effusion is assocated with air; for this reason a small amount of fluid usually escapes detection, while even in the presence of a large amount it is rarely possible to accurately map out its upper level. The dull note when present will be found to shift with the position of the patient. Though this movable dullness may at times be present in simple effusion, yet in no other disease is the phenomena so marked and constant as in pneumohydrothorax.

The signs obtained by auscultation are variable and misleading. In infants the auscultatory signs differ from those in adults

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The respiratory murmur is usually unchanged and succussion is seldom elicited. The metallic and amphoric qualities which the respiratory sounds possess in older children and adults are absent in infants. In older children the respiratory murmur is either

FIG. 8—RIGHT PYOPNEUMOTHORAX. VENTRODORSAL EXPOSURE. RECUMBENT POSTURE.

(E) Effusion.
(L) Partially collapsed lung adherent to posterior chest wall and unable to retract completely.
(H) Heart displaced to the left.
(Lv) Shadow of the liver merging with that of the effusion. Compare the costodiaphragmatic spaces. Note the presence of fluid in that of the right side.

entirely absent or feeble and distant. When the pneumothorax occurs in a previously healthy pleural cavity, and the lung is not diseased, the breath sounds may be harsh and loud. Whether

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feeble or intense, the respiratory murmur usually has an amphoric quality. This amphoric sound may be heard only over a certain part or over the entire affected chest. It is most frequently heard with expiration. In the intrascapular region the breathing may be tubular. The vocal resonance has a marked amphoric quality, and this may be present even when the respiratory murmur is absent. The so-called "metallic tinkle," which is really a râle with its pitch raised by the resonanting air chamber of the pneumothorax, may be heard at the end of expiration or during coughing. By using one coin as a plexor and one as a pleximeter, auscultation will reveal that the sound produced by the contact of the coins has been transformed into a clear musical sound, like the note of a bell. This phenomena, first described by Trausseau, has been called the "coin sound," and was considered pathognomonic of air in the pleura. Osler,[88] however, obtained this sound over a large cavity just underneath the pleura of the upper lobe. It cannot be denied that it is a sign of great value in children, in whom large cavitation of the lung is a rare condition. Over the fluid no respiratory sounds are obtained. By shaking the child, either in the recumbent or erect posture, the ancient sign "Succussio Hippocratis," a splashing sound may be heard if the amount of air associated with the fluid is not too small.

Examination with the Roentgen Ray.—The value of the Roentgen ray examination, in the study of the varied conditions present in pneumothorax, cannot be overestimated. It gives such accurate information concerning certain of the pathological conditions as is not obtainable in any other way. It makes possible the visual examination of the movements of the affected viscera and permits the systematic study of the progress of the disease. Valuable as this examination is, however, in furnishing important data, it should not be considered as a procedure which is to replace the older established methods of diagnosis, but rather is it to be held as a supplementary diagnostic method of undoubted accuracy. The difficulties which present themselves in the diagnosis of pneumothorax demand that all the diagnostic means at our disposal be employed in the effort to obtain a true idea of the conditions existing in this disease.

The examination may be made either with the aid of the fluorescent screen (Roentgenoscopy), which permits the study of the movements of the thoracic viscera, or a record of the pathological

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conditions may be obtained upon the photographic plate (Roentgenography). Both methods are to be employed, and the examination cannot be considered complete without either. For no description, however accurate, of the fleeting image seen upon the fluorescent screen can compare as a graphic record with the permanent impression on a photographic plate, which also gives the finer structural detail not to be seen with the screen. The picture of pneumothorax is so striking and characteristic that once seen the condition is thereafter easily recognized.

For the clear understanding of the conditions within the affected chest, information is necessary regarding the following points, upon which the Roentgen ray examination gives the required data:—

First.—Degree of Pneumothorax.—Has the air invaded the entire cavity or is it confined by adhesions to a particular part of the chest? The shadow of the normal lung as compared to that cast by the spine and mediastinal structures is bright, because the ray penetrates the air-containing tissues with greater ease than it does the dense structures. So also the shadow grows brighter during inspiration. With respiration, the movement of the normal lung shadows are visible. When air is present in the pleural cavity, however, the whole one part of the chest will be brilliantly illuminated. The ray will so affect the screen or plate as if nothing had been interposed in its course. The contrast between the part thus illuminated and the central dense structures is now very marked. Closer examination of this brilliant area will show that the normal lung markings are absent, while midway in the chest and against the spinal column an irregularly oval shadow with a well-defined outline will be noted. This shadow may enlarge and grow lighter during one or the other phases of respiration. The brilliantly illuminated part of the chest is that in which the pleural cavity contains air, while the mobile shadow against the spinal column is the more or less collapsed lung. Because of this collapse the penetration of the ray is facilitated, thus accounting for the brightness of the affected chest. The lattice-work effect of the ribs over the bright area is very clear, very distinct and of a lighter shade than the ribs on the normal side, while the structural detail of the ribs over this bright area are more clearly visible than on the normal side. The costo-diaphragmatic space is sharply defined and should always be examined for the presence of effu-

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sion. When, as in Figures 1, 4, 10 and 11, the air occupies every part of the pleural cavity, this characteristic appearance is present over the entire side, while if the air is localized only a circumscribed part of the chest will give the picture. The only other appearance which may simulate that of a localized pneumothorax is a cavity in the lung. In the latter condition, however, the con-

FIG. 9—PNEUMOTHORAX RIGHT. DORSAVENTRAL VIEW. (PLATE TO STERNUM.)

(L) Collapsed lung.
(A) Air in pleural cavity.
(D) Diaphragm.
(H) Heart displaced considerably to the left.
(S) Stomach—distended by gas.

trast between the normal and abnormal tissue is not so marked and the lighter area indicating the lung cavity is surrounded by a distinct ring of dense shadow corresponding to the cavity wall. Examination by the oblique method will also be of value in this differentiation.

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Second.—Condition of the Lung.—Is the lung normal or diseased? Is it completely or only partially collapsed? Tuberculosis, pneumonia and abscess are all demonstrable. When the lung is completely collapsed it may be impossible to definitely demonstrate the diffuse infiltration of a tuberculosis, but in such a case evi-

FIG. 10—Pneumothorax. To show the collapsed lung (L) lying as a ribbon-like mass along the spinal column. Note the distinctness of the ribs of that part of the pleural cavity (A) containing but air. (CS) Indicates the costo-diaphragmatic sinus.

FIG. 11—Pneumothorax. To show a collapsed lung which still contains considerable air and which was observed to distend and shrink with respiratory movements.

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dences of this disease will usually be found in the other lung. A large area of consolidation will not permit the lung to collapse to a marked extent. Occasionally the clue to the pathological primary process is derived from the condition of a part of the lung adherent, and therefore unable to retract. The lobes of the collapsed lung may be differentiated (Fig. 4), and in some cases one or other of the lobes not only contains more air, but distends to a greater extent during respiration. It is perhaps not an unjustified assumption that the more collapsed and more immobile lobe bears the perforation.

Third.—Position and Mobility of the Lung.—What is the position of the retracted lung? Does it functionate? If free to collapse, the lung is always found lying at its root. For some time after the perforation has occurred, the collapse is not complete. The lung still contains considerable air and casts a distinct shadow of considerable extent with a sharply defined border. When the causes which make for restitution are not at play, then the air in the collapsed lung is gradually absorbed; and as this progresses, the lung continues to shrink, so that in old cases it is to be seen lying airless and motionless, a ribbon-like mass, along the spinal column, as in Figure 10.

In closed pneumothorax, when the lung takes an active part in respiration, its appearance is as shown in Figures 4, 9 and 11. In these cases the lung may be observed during respiratory movements to expand from its shrunken state, distend so as to almost completely fill the chest, and then retract to its former partially collapsed condition at its root. Under certain conditions this movement of the partially collapsed lung is what is termed paradoxical; that is to say, instead of dilating with inspiration, the lung is inflated during expiration, and it is this expiratory force derived from the intact emphysematous (compensatory) lung which is the important factor in the restitution of conditions to the normal.

Fourth.—Condition of the Pleura.—Does it contain effusion? The difficulty of detecting by the usual diagnostic means, either the presence or the amount of effusion within a pleural cavity containing air, is well known to every clinician. With the aid of the Roentgen ray the smallest effusion may be detected. The costodiaphragmatic space should first be examined, and if this is clear there is no free effusion. The picture of the chest in

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hydropneumothorax will show three zones of shadow which differ in the intensity of their illumination. (Fig. 2.) There is a bright upper zone, a brilliant middle zone, and a dark lower zone. The bright upper zone corresponds to a part of the collapsed lung retracted and pressed against the upper and posterior part of the chest. The middle brilliant zone represents the part of the chest containing air. The contrast between this zone and the dark zone beneath it is very marked. This lower zone represents the fluid. It is a dense homogeneous shadow which may obscure the shadow of the ribs. Below it merges without any defined boundary into the shadow of the liver, while its upper level is sharply defined and perfectly horizontal. This upper level is sharply defined no matter how small the quantity of air. In simple effusion, on the other hand, there is no sharp line of demarcation at the level of the fluid, but the shadow of the latter gradually merges into that of the compressed lung. This level of the fluid in hydropneumothorax remains horizontal whatever the position of the thorax. When the effusion occurs late, that is, after the collapsed lung has become airless, or if the condition is an old one, then but two zones will be seen, an upper brilliant zone corresponding to the air and a lower dark zone corresponding to the fluid. The lung in these cases lies against the spinal column and its shadow merges with that of the mediastinum. This is the picture when the patient is examined in the upright position. With children, however, it is usually neither feasible nor advisable to examine them in the erect posture, and almost impossible to Roentgenograph them in this position. But when lying down, in which position they are best examined, the fluid flows over the posterior wall of the chest, while the air rises above it. The picture now obtained is a little more difficult of interpretation. (Fig. 3 and Fig. 5.) Instead of two sharply contrasted zones indicating the air and fluid, the entire chest is obscured by a light shadow, and it is only at the sides that the brilliant illumination due to the air is visible. The lung, however, still holds its position at the root. The motion of the fluid in response to the movements of the chest, diaphragm and heart has already been discussed. The parodoxical movement of the fluid may be studied. So passive is the diaphragm in some of these cases that sudden pressure upon the abdomen will cause a rise of the level of the fluid.

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